ECHOCARDIOGRAPHY : A CLINICAL APPRAISAL

Thesis presented to the University of Glasgow

for the degree of Doctor of Medicine

by

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J. Christine Rodger

INTRODUCTION

Currently there is considerable interest in non-invasive methods of cardiac investigation. Because echocardiography provides direct rather than indirect information on cardiac structure and function, it has important advantages over other methods. This thesis, based on three years' experience, is an account of my evaluation of echocardiography in a clinical context.

I first saw the method used in 1969 when I was working in the Department of Clinical Cardiology at the Royal Postgraduate Medical School. Echocardiography was then new to the United Kingdom and was generally regarded as far from respectable. Although its non-invasiveness was an obvious attribute, I became convinced of the fundamental importance of cardiac ultrasound when through it, my concept of the mechanics of the normal mitral valve was clarified.

I started using the method in 1971 when I first had access to ultrasonic equipment in the Department of Diagnostic Radiology in the Western Infirmary, Glasgow: my work has been done in and with the co-operation of that department. After an initial six month period spent overcoming technical problems and problems of echo identification, my colleague and I established a routine service in cardiac ultrasound. At first I was the only source of referral and even now, up to a third of the patients are examined at my instigation or request. Nonetheless, increasing interest in the method has been reflected in rising referral figures: 250 patients were examined in the last six months.

Although many of our early problems were due to the intrinsic deficiencies of our original recording systems and would have been avoided had a strip-chart recorder been available, the lack of detailed published information on methods of examination, recording and measurement undoubtedly also played a part. These considerations go a long way towards explaining why Feigenbaum (1973) has found that much of the echocardiography done today is totally inadequate.

As my experience broadened, I became increasingly critical of the quality

and/...

and interpretation of published echograms. I realised too that because there had been insufficient emphasis on methods of measurement and the reproducibility of results, the quantitative aspects of cardiac ultrasound were suspect. I concluded that a number of facets of echocardiography required further critical appraisal. I therefore compiled and analysed the data set out here to provide a background for research and for routine clinical investigation. Unfortunately, parallel haemodynamic studies were often impossible: my work was done in a hospital in which I was not based and I did not have access to the necessary facilities.

This thesis is basically an evaluation of the accuracy and limitations of cardiac ultrasound. However, I have made some use of echocardiography as a research tool e.g. in the assessment of left ventricular function in chronic renal failure and the results of this approach are also presented.

New prospects for cardiovascular research have been opened up by single dimensional and more recently multidimensional echocardiography. It may indeed be true that echocardiography has reached a stage of its development comparable to that of electrocardiography 30 years ago (British Medical Journal, 1974). If so, its potential is enormous.

KEY TO FIGURES AND TABLES

CARDIAC STRUCTURES

In the ultrasonic tracings and the diagrams cardiac structures have been identified as follows:-

AC	mitral anterior cusp
PC	mitral posterior cusp
R	mitral ring
Ch	chordae tendineae
A Ch	anterior chordae
P Ch	posterior chordae
PM	papillary muscle
PPM	posterior papillary muscle
RV	right ventricle
LA	left atrium
LV	left ventricle
endo	left ventricular endocardium
epi	left ventricular epicardium
myo	left ventricular myocardium
IVS	interventricular septum
Me	membranous portion of interventricular septum
Mu	muscular portion of interventricular septum
Ao	aortic root
T	tricuspid valve
PE	pericardial effusion

The following abbreviations have been used in the tables and graphs:-

AMI	acute myocardial infarction
AS	aortic stenosis
AR	aortic regurgitation
Ca	calcification
Co	cardiac output
CTR	cardio-thoracic ratio
CXR	chest x-ray
DCR	diastolic closure rate of the mitral valve
EDV	left ventricular end-diastolic volume
ESV	left ventricular end-systolic volume
EF	left ventricular ejection fraction
LVEDP	left ventricular end-diastolic pressure
LVF	left ventricular failure
MS	mitral stenosis
MR	mitral regurgitation
PCV	packed cell volume
PWDV	left ventricular posterior wall diastolic velocity
PWE	left ventricular posterior wall excursion
RDT	regular dialysis therapy
SV	left ventricular stroke volume

CHAPTER I

EQUIPMENT

Professor Ian Donald has pioneered the application of ultrasound to medicine (Donald and Brown, 1961). Engineers from his department of ultrasonics technology are responsible for the maintenance and development of the equipment which we use for echocardiography. Because part of their remit as experts is the evaluation of new and prototype equipment, we have the opportunity to try-out new ultrasonic units and recording systems before they become generally available. The apparatus which we use routinely at present is the Eskoline 20 ultrasonoscope, interfaced with an ultraviolet strip-chart recorder (Honeywell Visicorder).

Eskoline 20 Ultrasonoscope

This apparatus displays echocardiographic information in the 'A' (amplitude modulated) or 'TP' (time-position) modes.

A scan display

The A scan is used to locate intra-cardiac structures. Echoes are displayed as vertical displacements from a horizontal axis (Fig. 1,1). The amplitude of the echoes received from a structure is a function of its reflectivity and of its depth from the transducer. Thus, the amplitude of the echoes received from structures of equal reflectivity but at different depths, varies. To compensate for the depth component a control voltage, the time-varied or swept gain, is applied to the receiver amplifier. The characteristics of the voltage waveform are such that amplifier gain is low over the first few centimetres and increases with depth from the transducer. Fig. 1,2 shows the effect of varying swept gain waveform on the amplitude of the anterior echoes in a string of exponentially decaying echoes.

Time-position (TP) display.

Echocardiographic records are made from the time-position display. The echoes are displayed as dots along an invisible horizontal timebase. A marker grid is produced by intensifying the timebase at 1 cm. intervals and slowly sweeping the timebase upwards (Fig. 1,3). The A scan echoes are applied to the cathode/...

cathode ray tube to intensify the timebase : varying echo strength is expressed as varying dot intensity and movement of the echoes with time is displayed by slowly sweeping the horizontal timebase upwards. Echoes recorded from moving and static structures using this technique are shown in Fig. 1,4.

The echoes on the A scan vary in amplitude (Fig. 1,1) : a major disadvantage of the Eskoline 20 is that these signals are used directly to intensify the display in the TP mode. The dots which they produce on the TP display vary in length and there is resulting loss of information. In more sophisticated processing systems, fixed amplitude signals are generated and resolution is improved (Fig. 1,5).

Recording systems.

Ideally, only a system which can provide a continuous record of multiple echoes should be used for echocardiography. The following considerations explain why two of the three recording systems which we have used were basically unsatisfactory.

To obtain a valid echogram from an intra-cardiac structure, the incident ultrasonic beam must strike it at or near right angles. This requirement is only fulfilled intermittently because respiratory movement and minor alterations in the angulation of the hand-held transducer change the direction of the beam. At any examination some of the recorded echocardiographic complexes are therefore invalid and a number of complexes must be recorded before accurate measurement is possible. To use ultrasound to measure the dimensions of a cardiac chamber or to display the anatomical relationships between structures, multiple echoes must be recorded.

1) Polaroid photography.

The time-position display is recorded directly from the face of the oscilloscope with a Polaroid camera. Multiple echoes are recorded but the record is not continuous. The method has a number of disadvantages.

Because each record comprises a single oscilloscope sweep, at normal heart rates no more than 6 consecutive cardiac cycles can be recorded and the time spent in/...

in obtaining valid echocardiographic information can be considerable. Camera settings require to be changed when the sweep speed is changed and also when electronic drif alters the trace brightness. Astigmatism of the cathode ray tube produces distortion of the marker grid (Fig. 1,3) and of the recorded waveform. Thus, this method of recording was found to be time-consuming. Further, using it there was difficulty in making accurate measurements and in inter-relating echoes from different intra-cardiac structures.

2) Analogue gate with paper chart recorder.

An analogue gate was used to interface the Eskoline 20 with a paper chart recorder (Mingograf 31B). The gate produces a DC signal which drives the chart recorder. It is positioned on the A mode display so that it encompasses the maximum movement of the echo to be recorded. The voltage of the signal from the gate varies with the movement of the echo within it.

Although it provides a continuous tracing, this system has the serious disadvantage that only one echo can be recorded. Further, distorted waveforms are recorded if the echo moves out of the gate, if another echo enters it or if echo drop-out occurs (Fig. 1,6).

We have thus found the method to be unsatisfactory and have never used it on a routine basis.

3) Ultraviolet strip-chart recorder.

The development of fibre-optic faceplates has made it possible to obtain an in-focus image on the outside face of a cathode ray tube. The focused image can be transferred to ultraviolet sensitive paper placed in contact with the outside face of the tube and a continuous record can be made by drawing the paper across the surface of the tube. The Honeywell Visicorder and the Medelec M scope are two commercially available recording systems which utilise these principles. They are ideal for echocardiography because they can provide a continuous record of multiple echoes. The paper width and the spot size of the tube were among the considerations on which our decision to use the Honeywell Visicorder was based. It was interfaced with/...

with the Eskoline 20. The marker system was modified to provide 2 mm depth markers at accurate, mains-referenced intervals of 2 sec. Video-blanking was used during the marker write-out to ensure that the markers were clearly visible in areas of high echo-density.

We have used this recording system for a year. With it, not only has the time required for each examination been considerably reduced but echo identification and the accuracy of measurements have improved.

The rate of progress of echocardiography has reflected the rate of progress of its technology. That echocardiography is now in a phase of rapid development can be ascribed in part to the introduction of ultraviolet strip-chart recording systems. Similarly, the serious disadvantages of previous recording systems undoubtedly retarded the development of the method over a number of years.

Now that it is possible to make continuous records of detailed echocardiographic information, standardisation of instrumentation and of recording methods should be achieved.

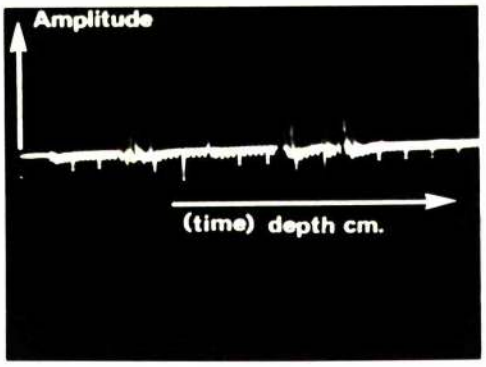
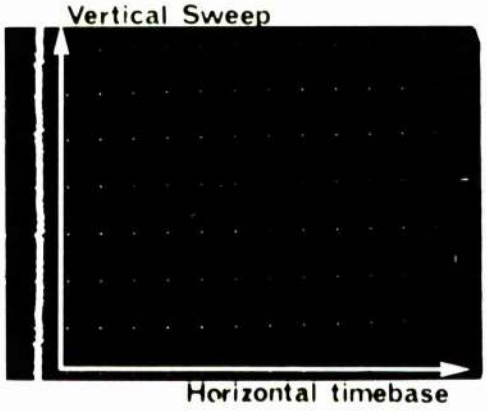


FIG. 1,1 A SCAN DISPLAY



TP DISPLAY - MARKER GRID

FIG. 1,3

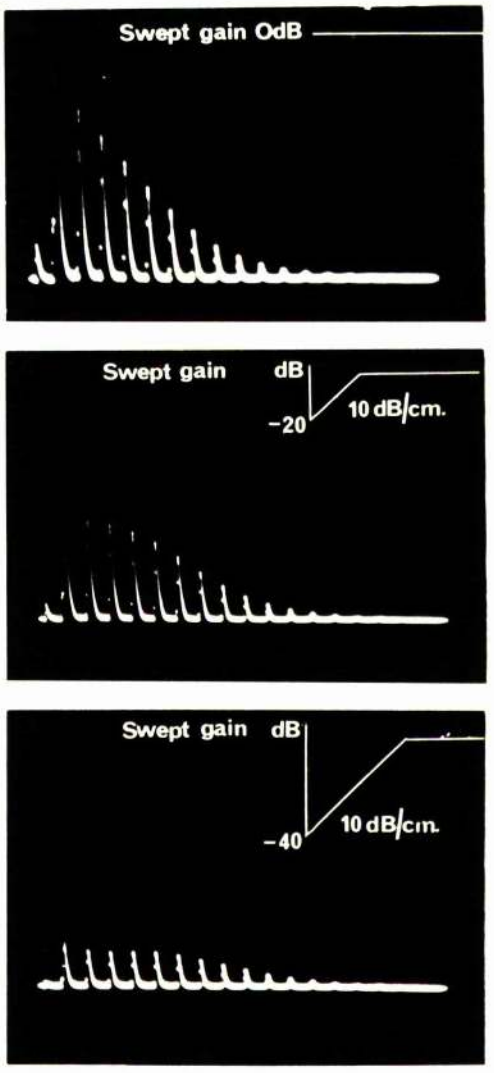


FIG. 1,2 EFFECT OF VARYING SWEPT GAIN WAVEFORM

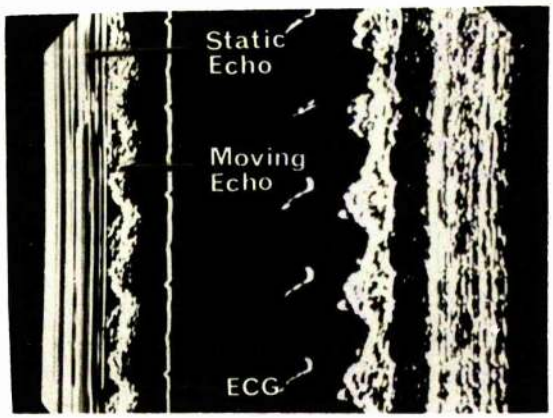


FIG. 1,4 TP DISPLAY

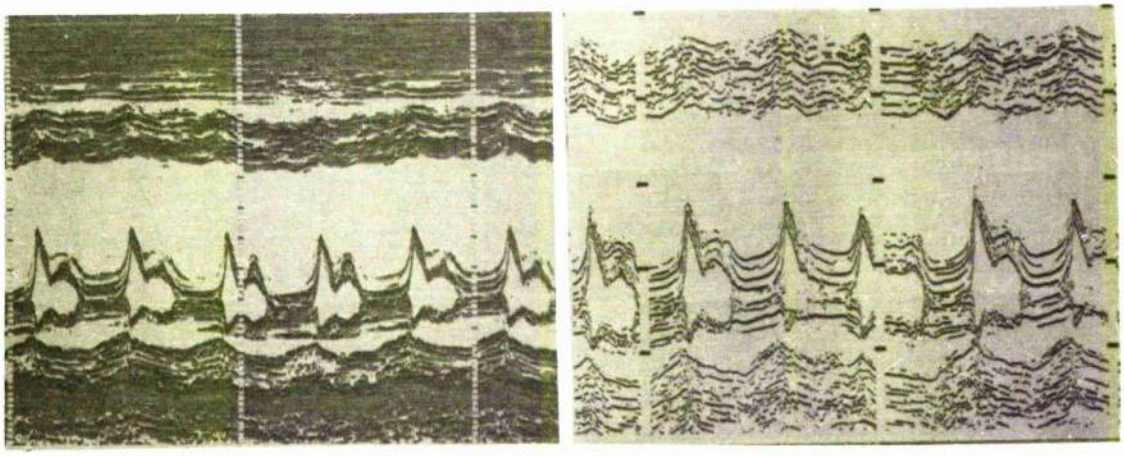


FIG. 1,5 (a) ESKOLINE 20 (b) N.E. DIASONOSCOPE

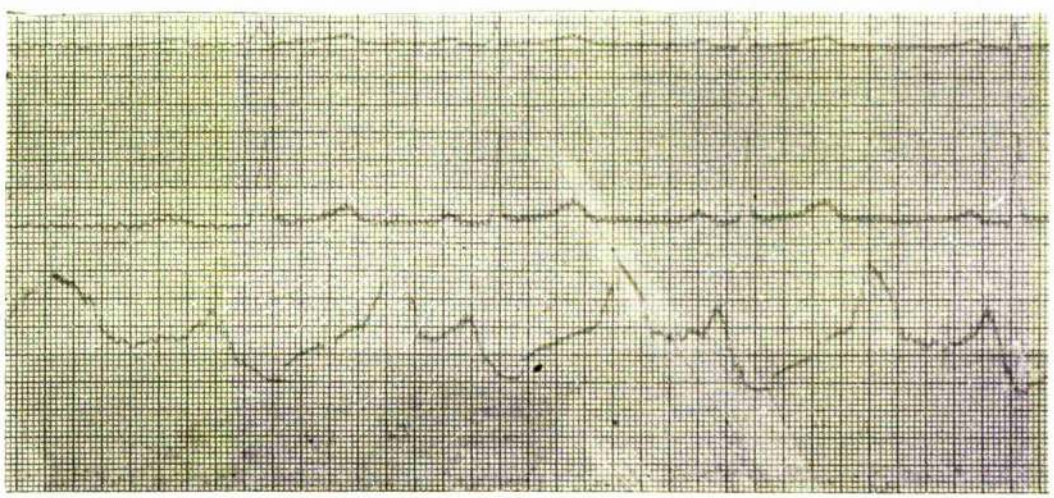


FIG. 1,6 ANALOGUE GATE SYSTEM RECORD

MEASUREMENT OF THE DIASTOLIC CLOSURE RATE OF THE NORMAL MITRAL VALVE

Although echocardiography is being increasingly accepted as a valid method of cardiac investigation, instrumentation and recording techniques are not yet standardised (Joyner, 1972). No standard methods of measurement from recorded echograms have been described and the within-subject variation of such measurements has not been assessed.

Edler (1961) was the first to measure the diastolic closure rate of the mitral valve from the anterior cusp echogram in normal subjects. Published values for the diastolic closure rate of the normal mitral valve vary (Table 1). This variation, which undoubtedly reflects differences in methods of recording, may also reflect differences in methods of measurement.

There is general agreement on the diastolic closure rate of the rheumatic mitral valve: the information obtained from this measurement is clinically useful and has been well substantiated (Effert et al., 1964; Segal, Likoff and Kingsley, 1966; Edler, 1967; Winters et al., 1967). In non-rheumatic mitral regurgitation (Segal, Likoff and Kingsley, 1967; Sweatman et al., 1972; Tallury, De Pasquale and Burch, 1972,; Millward, McLaurin and Craige, 1973) and in congestive cardiomyopathy (Ziady et al., 1973; Millward et al., 1973; Layton et al., 1973), the published mitral valve diastolic closure rates are quite widely discrepant. Until all echocardiographic records and the methods of measurement from them are comparable, studies of closure rates by different groups of workers will continue to produce confusing results.

The mitral valve diastolic closure rate, considered in conjunction with other echocardiographic and haemodynamic measurements, may provide an index of left ventricular filling (Layton et al., 1973). Before this suggestion can be validated, more precise and reproducible measurement of the diastolic closure rate is necessary.

This report describes how the form of the diastolic closure slope recorded from a normal mitral valve can vary. It indicates to what extent this variation alters/...

alters the diastolic closure rate measured from the slope and suggests a standard method of measurement.

Subjects and Methods

Forty-five normal subjects and one with lone atrial fibrillation were studied. Their ages ranged from 15 to 50 (mean 29) years and 32 were female.

The mitral anterior cusp echogram was recorded with the subjects supine and care was taken to record the maximum amplitude of mitral movement.

The apparatus used was an Eskoline 20 ultrasonoscope (Smith Kline Instruments) with an unfocused transducer. In 45 subjects, records were made directly from the oscilloscope with a Polaroid camera. In the latter part of the study an ultraviolet strip chart recorder (Honeywell Visicorder) was available and with it records were made in 5 subjects at chart speeds of 10 and 20 mm/sec and in one subject, at speeds up to 100 mm/sec. On the Polaroid records, vertical markers indicating one cm. of tissue depth were recorded at intervals of 0.5 sec. and on the strip chart records, 0.2 cm. depth markers were recorded at intervals of 2 sec.

On the strip chart records of each of 5 subjects, the diastolic closure rate was measured from mitral echograms in which the form of the diastolic closure slope varied. Selection criteria, which allowed reproducible closure rate measurements, were established. Using only complexes which fulfilled these criteria, within-subject variation was assessed from chart recordings in two normal subjects and one with lone atrial fibrillation, while between-subject variation was investigated from the Polaroid records of 45 normal subjects.

Results

Variation in diastolic closure slope and measured diastolic closure rate.

The echograms in Fig. 2,1 were recorded from the same subject at a single examination. The amplitude, the form of the diastolic closure slope and the diastolic closure rate measured from the slope vary. In record 4 (Fig. 2,1), the amplitude is maximal and diastolic closure is monophasic, i.e. has a single component. In records 1 to 3 (Fig. 2,1), the amplitude of all complexes is submaximal and diastolic closure is basically biphasic, i.e. has two main components/...

components, the first of which is relatively slow. The duration and speed of each of the two components vary : for the second component, this is well illustrated in record 3 (Fig. 2,1).

Fig. 2,2, recorded from another subject, also illustrates the variability of the diastolic closure rate measured from complexes in which the form of the diastolic closure slope differs. Complexes 1, 2 and 8 are monophasic and the measured diastolic closure rates are in close agreement.

A standard method of measurement of the diastolic closure rate.

From Figs. 2,1 and 2,2 it can be seen that measurement of the diastolic closure rate from a closure movement with a sirgle component is technically easy and provides reproducible results. It is evident that when there is more than one component, a decision on which of these to measure must be made. If, for a biphasic closure movement, the initial component is chosen, the measured diastolic closure rate is slow, and if the second component is measured, the diastolic closure rate is fast. Further as is illustrated in Fig. 2,1 record 3, although measurement of the fast component of a biphasic closure movement may often produce a diastolic closure rate comparable to that measured from a monophasic one, more variation can occur.

It is always possible to record a monophasic diastolic closure movement from a normal mitral valve, (Fig. 2,3). In Figs. 2,1 and 2,2 the complexes with monophasic movements have a maximal amplitude.

It is concluded that when the amplitude of the recorded echogram is maximal, diastolic closure of the normal mitral valve is always monophasic. It is therefore suggested that to standardise results, measurement of the diastolic closure rate of the normal valve should be made only from mitral anterior cusp echograms in which a single component diastolic closure movement has been recorded.

Within-subject variation of the diastolic closure rate.

To investigate within-subject variation of the diastolic closure rate, only echograms in which the closure was monophasic were selected for measurement. The diastolic closure rates of a series of complexes were measured on strip chart records from/...

from each of 3 subjects, two normals and one with lone atrial fibrillation.

Forty-five complexes were measured in one subject and the distribution of the closure rates obtained is shown in Fig. 2,4. The range is 130 to 210 mm/sec, the mean 160 mm/sec. and the standard deviation 14 mm/sec. The curve superimposed is the normal distribution for this mean and standard deviation. The data are a good fit to a normal distribution. This suggests that the within-subject variations are random and that no systematic bias has been introduced by the selection criteria.

Measurements were made from 15 complexes in the other normal subject and from 13 complexes in the patient with lone atrial fibrillation. The means and standard deviations for these two series of measurements were 201$\pm$ 11 mm/sec and 200$\pm$ 10 mm/sec respectively : the standard deviations are comparable to that of the series in Fig. 2,4. In atrial fibrillation, therefore, the varying ventricular rate does not affect the mitral diastolic closure rate : this can be confirmed by inspection of Fig. 2,5. Fig. 2,6 shows that in resting normal subjects in sinus rhythm, the diastolic closure rate and the heart rate are unrelated.

The normal mitral diastolic closure rate.

To investigate between-subject variation only echograms with a monophasic diastolic closure movement were selected and the diastolic closure rate was measured from the Polaroid records of 45 normal subjects. At least 2, and up to 6, complexes were measured in all subjects and a mean diastolic closure rate was calculated for each. The standard deviations were comparable to those obtained from the 3 subjects in whom a larger series of measurements was made. The mean values for the 45 subjects are shown in Fig. 2,7. The range is 128 to 234 mm/sec with a mean of 176 mm/sec. and a standard deviation of 29 mm/sec. When these results are compared with those obtained from multiple measurements in a single subject (Fig. 2,4), it is evident that the ranges are comparable. However, the values from the group of 45 subjects are more evenly distributed throughout the range and give a standard deviation of more than twice that of the within-subject study. The findings suggest that there is a real between-subject variation in the normal mitral diastolic closure rate.

Discussion/...

Discussion

The variation in published values for the diastolic closure rate of the normal mitral valve (Table 2,1) undoubtedly reflects differences in methods of recording : some of the workers used Polaroid records of the oscilloscope display and others, employing an analogue gate system, recorded a single echo on an ink or photographic recorder. Both these recording methods have disadvantages (Ross, 1972; Joynor, 1972) and echograms obtained by them are unlikely to be technically comparable.

The variation in published normal values may also reflect differences in methods of measurement : few of the authors detail their method of measurement, none comments on the variable form of the recorded diastolic closure movements and only one indicates the extent of the variation in calculated diastolic closure rates from a single subject. In echograms in which the closure movement has two components, Ross, (1972) measures the slower first component. Other authors illustrate diagramatically the method of measurement when closure is monophasic but fail to describe how the diastolic closure rate was calculated from their actual records which are often biphasic.

Strip chart records are a major advance. Because multiple echoes and an unlimited number of cardiac cycles can be recorded, the accuracy of measurements made from the echograms can be improved. From strip chart records it has been concluded firstly that monophasic diastolic closure movements can always be recorded from a normal mitral valve and secondly that when diastolic closure is monophasic the amplitude of the echogram is maximal, while when closure is biphasic the amplitude is submaximal. The form of the recorded closure movement is thus a function of the amplitude of the echogram.

It is technically easy to measure the diastolic closure rate from a monophasic record and this study has shown that the results are reproducible. It is suggested that to standardise measurement of the diastolic closure rate, measurements should be made only on echograms with a monophasic diastolic closure movement. These echograms have a maximal amplitude. Wilde and Pridie (1973) did not consider the diastolic/...

diastolic closure slope or diastolic closure rate but stated that for a reproducible mitral echogram the amplitude should be maximal. Selection for measurement according to the form of the diastolic closure movement has the practical advantage that suitable complexes can be identified on previously recorded traces. It would otherwise be impossible to decide which echograms on these traces were of maximal amplitude.

Although the diastolic closure rate should be measured on monophasic records, the relation between these measurements and haemodynamic events is uncertain. Because the echograms thus selected for measurement have a maximal amplitude, they are theoretically likely to derive from the free margin of the mitral anterior cusp. In Fig. 2,8 several echograms comprise both monophasic and biphasic closure movements : the origin of the monophasic movement is always anterior to that of the biphasic one, consistent with its derivation from the free edge of the leaflet. Inspection of Fig. 2,8, from a subject with systemic hypertension, also illustrates how, particularly when using an analogue gate system, the most anterior portion of the echogram may be omitted. In these circumstances a biphasic closure movement originating behind the free edge of the cusp,will be recorded.

Applying this standard method of measurement, the mitral diastolic closure rates recorded in normal subjects are higher than most of those previously published. Layton et al, (1973) used an analogue gate system and attributed the slow closure rates which they observed to the fact that they were recording at chart speeds of 100 mm/sec. However, in this present study records were made in a normal subject at speeds up to 100 mm/sec. (Fig. 2, 9) : it was found that with the probe positioned to give a monophasic closure movement at low speeds, an increase in chart speed introduced no additional components. Therefore, measurements of the diastolic closure rate at different recording speeds give consistent results.

Although it has been possible to make reproducible measurements of the diastolic closure rate and the standard deviation of within-subject measurements has been shown to be acceptably small, it is still possible to record widely varying closure rates from a single subject (Fig. 2,4). To estimate the diastolic closure rate/...

rate accurately it is thus essential to make measurements from a series of complexes. Because of the disadvantages of other recording methods, these measurements are best made on strip chart records. Measurement of monophasic closure movements on strip chart records should provide results of sufficient accuracy to allow investigation of the recent suggestion that the mitral diastolic closure rate can be used as an index of left ventricular filling (Leyton et al., 1973)

Summary

Published values for the diastolic closure rate of the normal mitral valve vary and reflect differences in methods of recording and measurement.

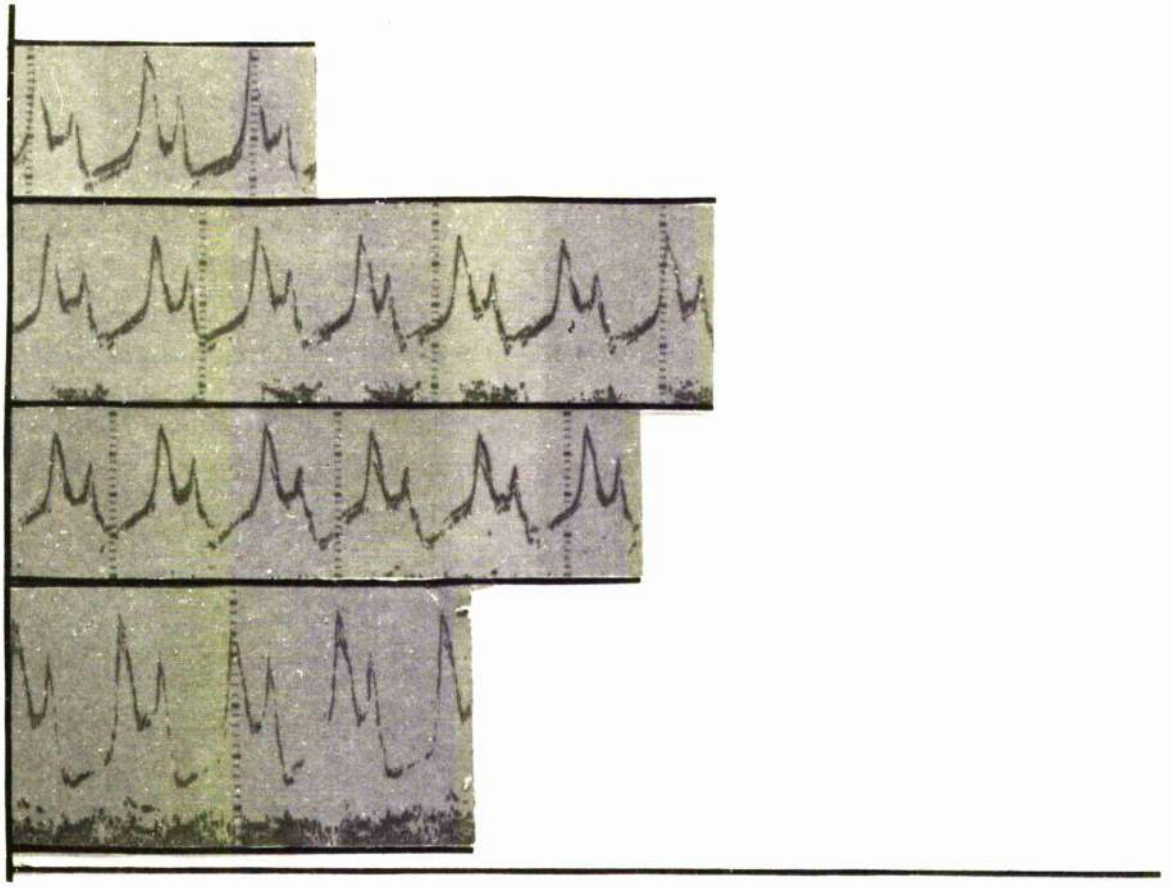
From strip chart records it was concluded that the form of the recorded mitral diastolic closure slope can vary, that reproducible measurements of the closure rate can be made from echograms with monophasic closure movements and that the amplitude of these echograms is maximal.

Measuring only complexes with monophasic closure movements, the within and the between-subject variations of the normal mitral diastolic closure rate were investigated. The ranges obtained from multiple measurements in a single subject and from a group of 45 normal subjects were comparable but the distribution of the results differed. It was concluded that there is a real between-subject variation in the normal mitral diastolic closure rate and that the diastolic closure rate in a single subject should be determined by measurement of a series of complexes.

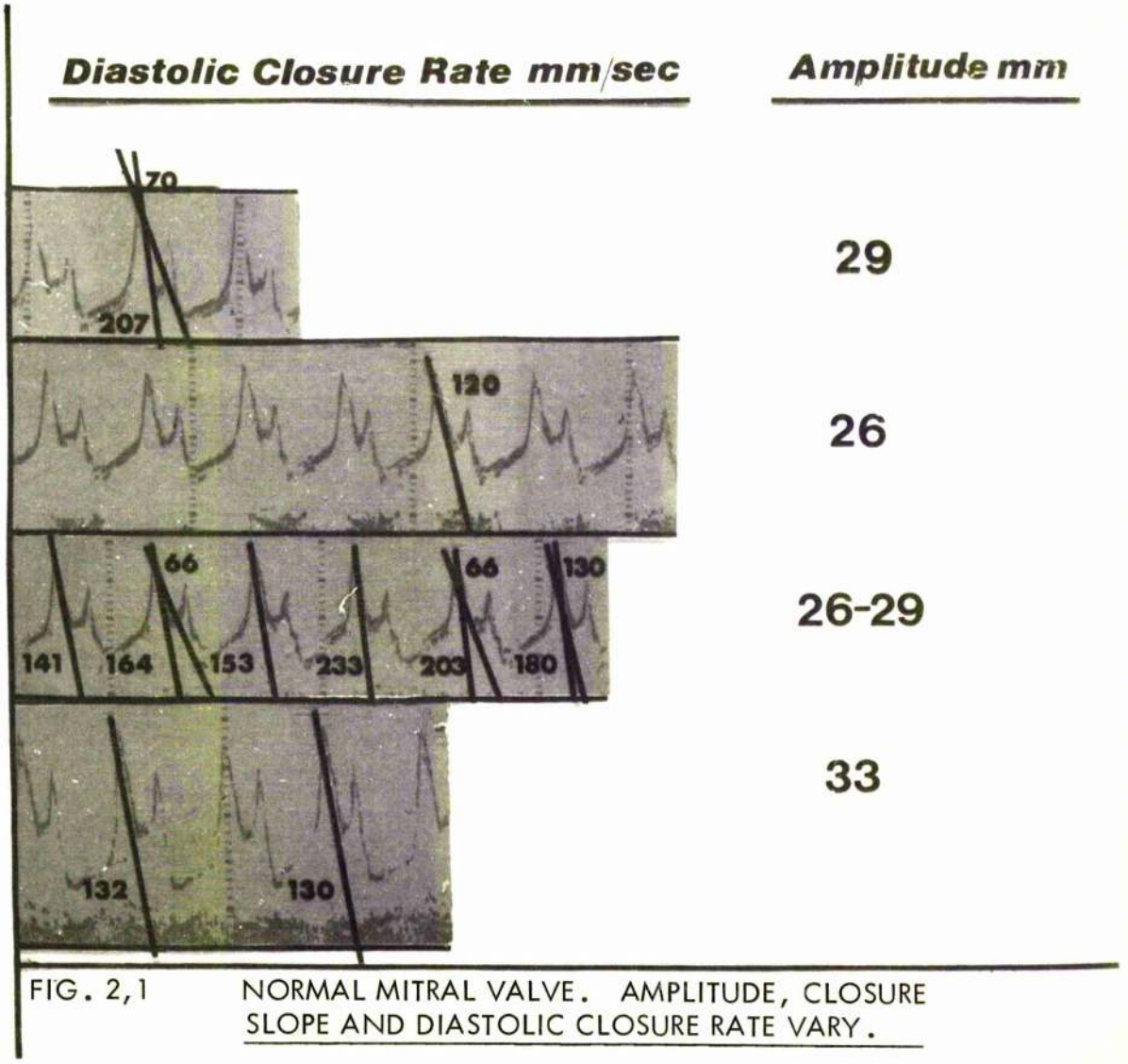
The accuracy of measurement of the diastolic closure rate of the normal mitral valve has been improved by using strip chart records and by measuring only echograms with a monophasic diastolic closure movement.

TABLE 2,1 PUBLISHED VALUES FOR THE DIASTOLIC CLOSURE RATE OF THE NORMAL MITRAL VALVE

	Number of subjects studied	Diastolic closure rate - range mm./sec.	Diastolic closure rate - mean mm./sec.
Edler, 1961	77	80 -	120
Joyner, Reid and Bond, 1963	25	85-160	
Effert et al., 1964		80-200	125
Segal, Likoff and Kingsley, 1966	50	70-150	
Edler, 1967	53	90-190	140
Winters et al., 1967	75	65-210	
Wharton and Lopez Bescos, 1970	12	80-150	123
Gramiak and Shah, 1971		80-150	
Pridie, Benhem and Oakley, 1971		70-120	
Ross, 1972	39	70-160	114
Ziady et al., 1973	46	120-200	
Layton et al., 1973	5	58-139	92

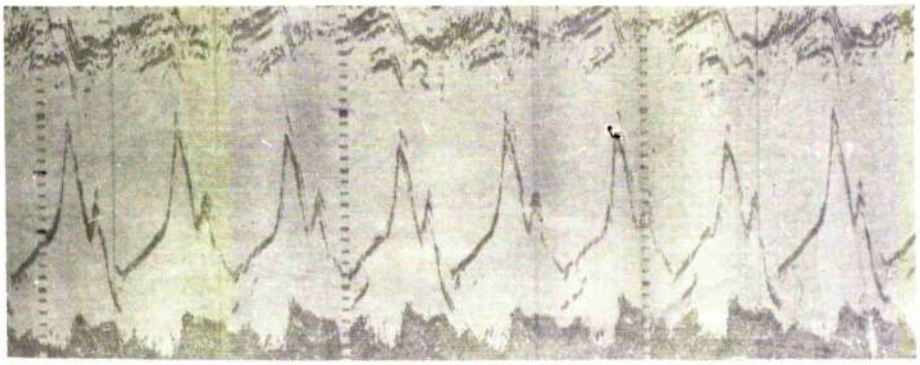


(a)



(b)

FIG. 2,1 NORMAL MITRAL VALVE. AMPLITUDE, CLOSURE SLOPE AND DIASTOLIC CLOSURE RATE VARY.



Diastolic Closure Rate mm/sec

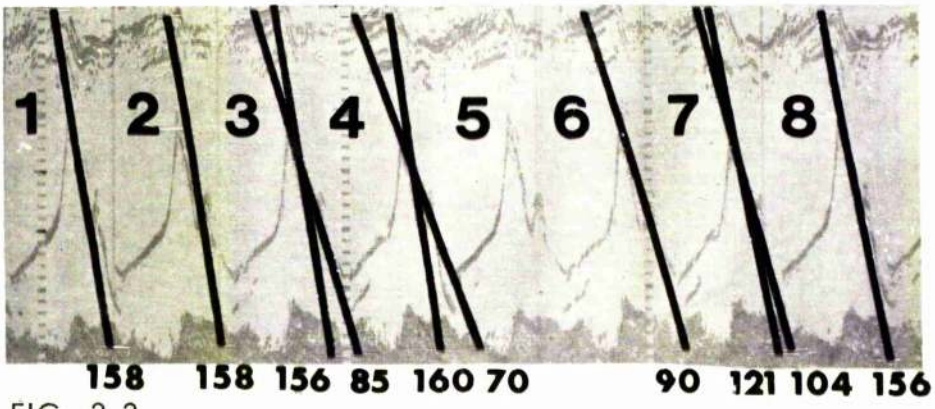


FIG. 2,2

DIASTOLIC CLOSURE IS MONOPHASIC IN COMPLEXES 1, 2 and 8 AND BIPHASIC IN 3, 4, 6 and 7.

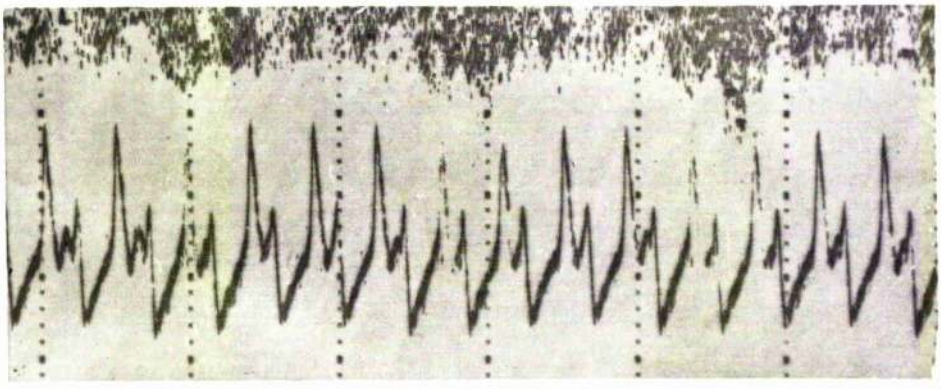


FIG. 2,3 DIASTOLIC CLOSURE IS MONOPHASIC

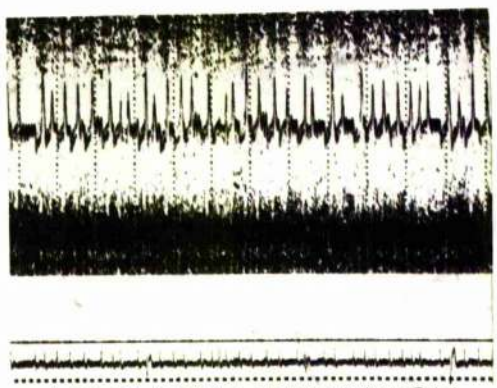


FIG. 2,5 LONE ATRIAL FIBRILLATION

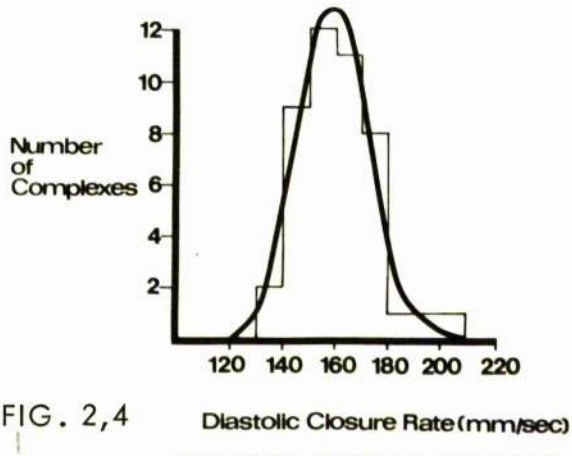


FIG. 2,4 DIASTOLIC CLOSURE RATE - WITHIN SUBJECT VARIATION

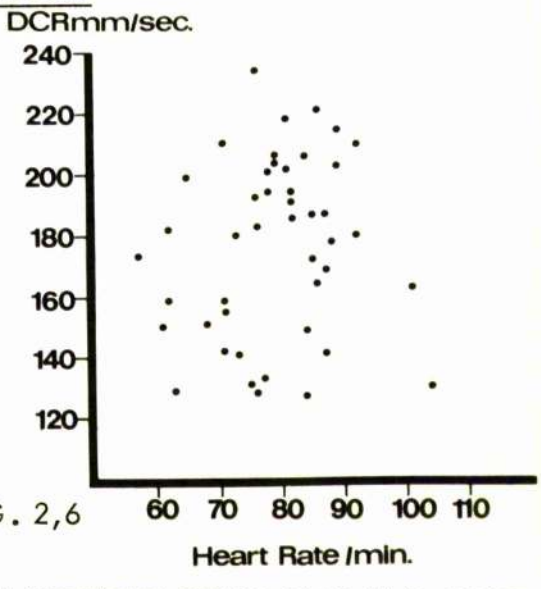


FIG. 2,6 DIASTOLIC CLOSURE RATE AND HEART RATE IN 45 NORMAL SUBJECTS

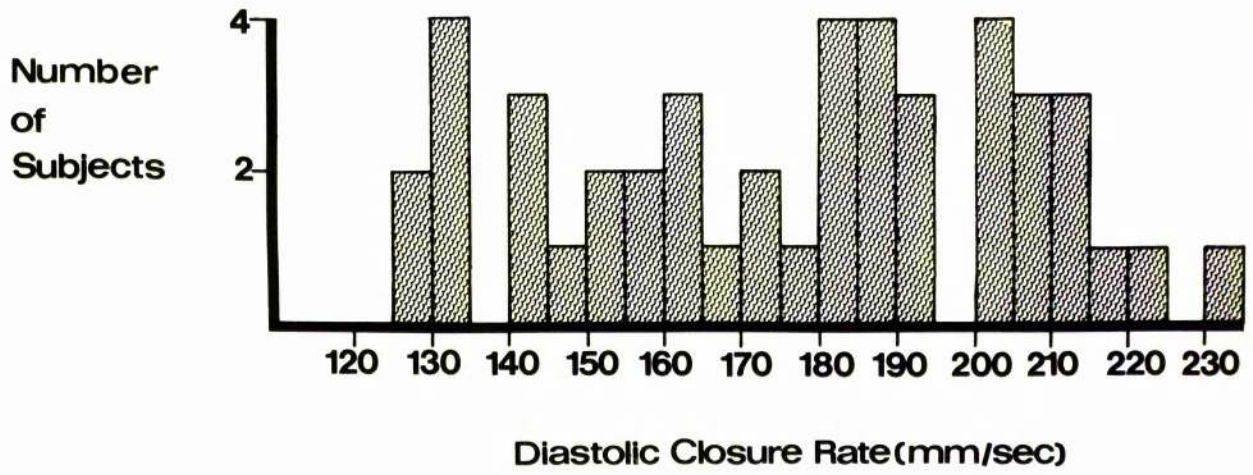


FIG. 2,7 DIASTOLIC CLOSURE RATE - BETWEEN SUBJECT VARIATION

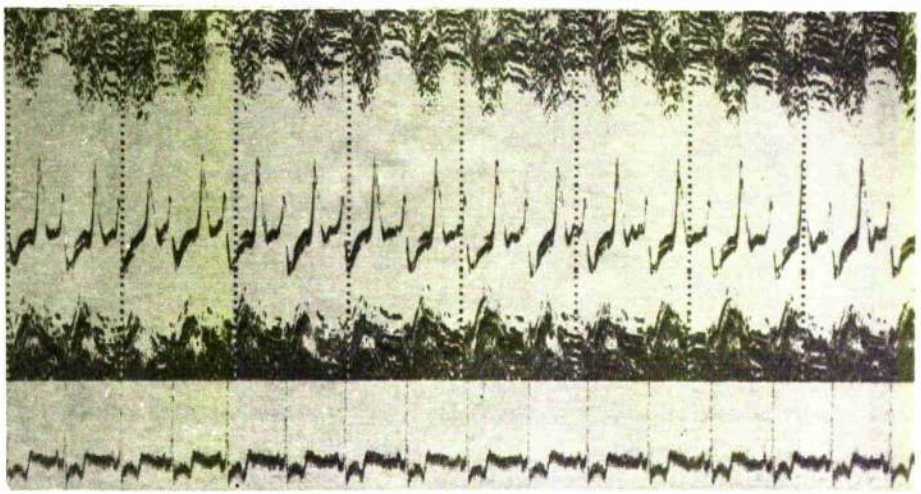
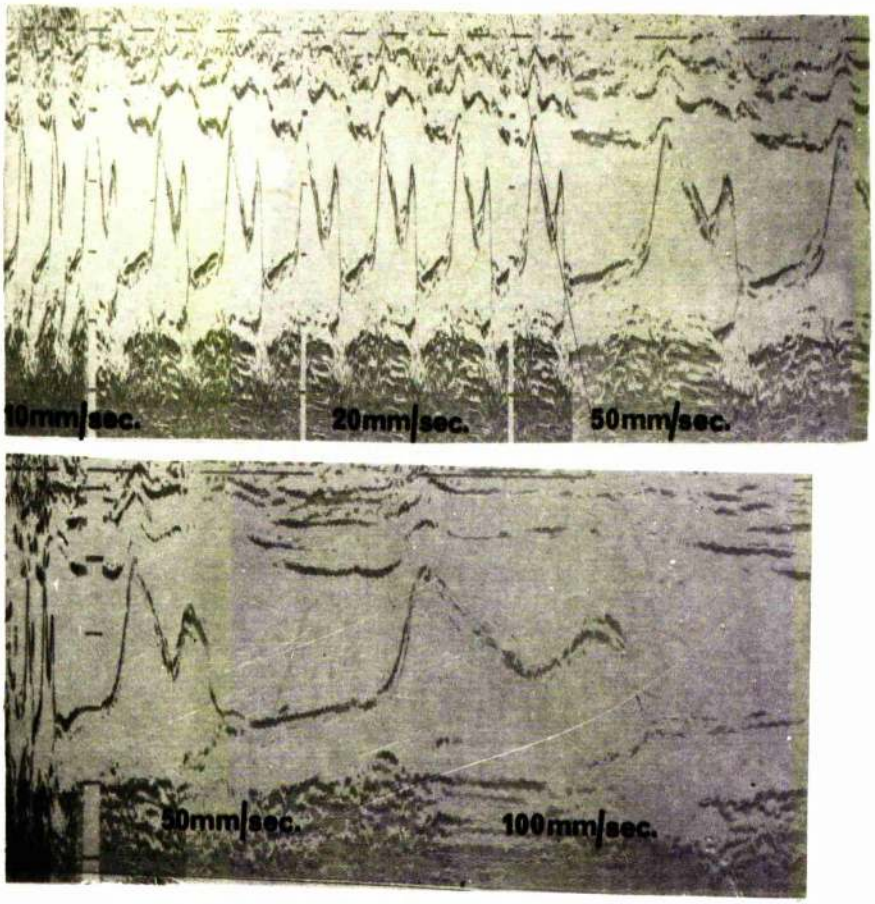


FIG. 2,8 MONOPHASIC CLOSURE MOVEMENTS ORIGINATE IN FRONT OF BIPHASIC ONES



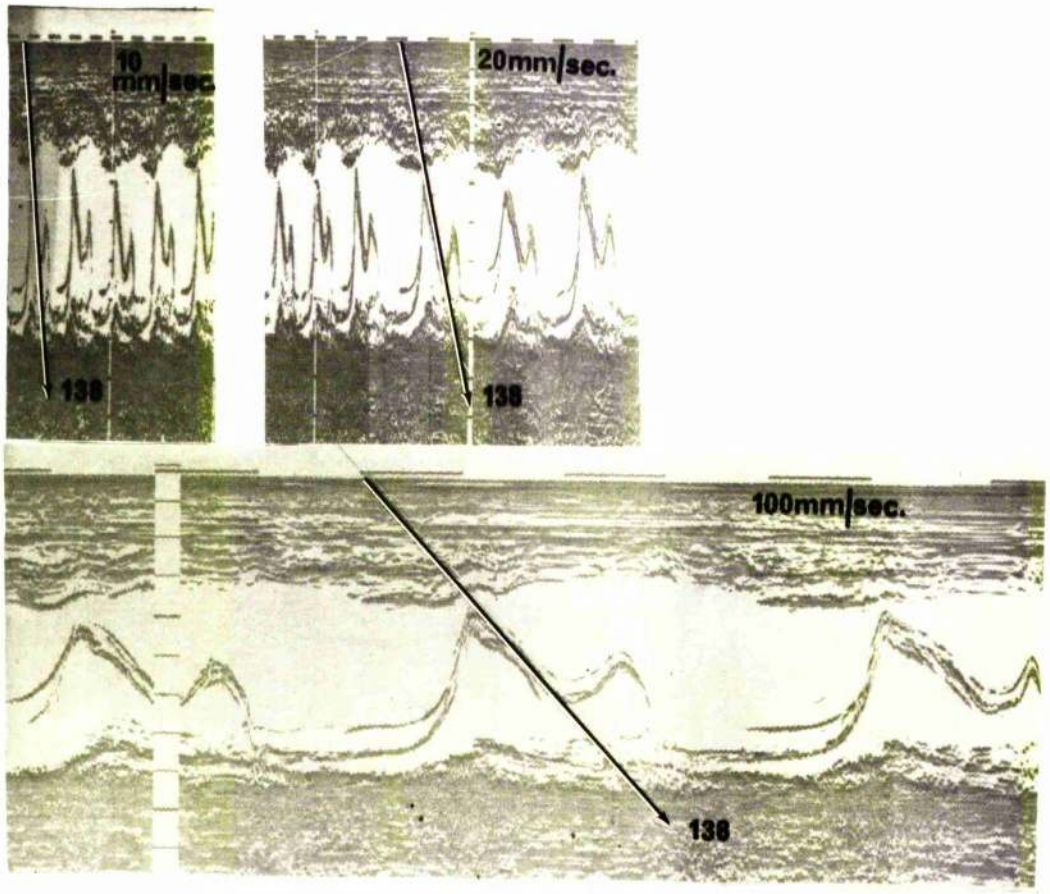


FIG. 2,9 DIASTOLIC CLOSURE AT DIFFERENT CHART SPEEDS

CHAPTER 3

THE MITRAL DIASTOLIC CLOSURE RATE IN MYOCARDIAL INFARCTION

The determinants of the diastolic closure rate of the mitral valve have not been established. Cardiac lesions characteristically associated with either an increased or a decreased diastolic closure rate have, however, been defined.

The mitral diastolic closure rate in the resting normal subject is unrelated to heart rate (Chap. 2). In hypertrophic obstructive cardiomyopathy, the left ventricle is resistant to filling and the mitral diastolic closure rate is reduced (Popp and Harrison, 1969). In non-rheumatic mitral regurgitation, an increased volume of blood is delivered to the left ventricle under an increased left atrial pressure and the mitral diastolic closure rate is increased (Segel, Likoff and Kingsley, 1966). These established associations suggest a relation between the mitral diastolic closure rate and left ventricular filling.

Left ventricular filling pressure has been shown to be consistently elevated in patients with acute myocardial infarction who have clear clinical evidence of left ventricular failure (Wolk, Scheidt, and Killip, 1972; Scheidt et al., 1973).

The basic aim of this clinical study was to measure the mitral diastolic closure rate in the same subjects in both the acute and the convalescent phases of myocardial infarction and thus to investigate the relation between the left ventricular filling pressure and the diastolic closure rate.

Patients

Mitral echograms were recorded within 48 hours of myocardial infarction in 55 unselected patients admitted to a coronary care unit. Technically satisfactory records were obtained in 40 and only these patients were included in the study. Their ages ranged from 35 to 80 (mean 56) years: 34 were males. All the patients had raised serum AsT and LDH levels; 36 had Q wave evidence of/...

of infarction and 4 showed sequential ST-T changes only. The infarct was anterior in 22 and inferior in 18 cases.

Six patients died in hospital and two died within a week of their discharge. Thus the hospital mortality for the group was 15% while the mortality at one month was 20%. Post-mortem examination was carried out in 5 cases.

Of the 26 patients available, 24 with technically valid echograms were included in the follow-up study. In 17, the time from infarction to follow-up was between 5 and 8 weeks: the longest interval before follow-up examination was 7 months.

Methods

The apparatus used was an Eskoline 20 ultrasonoscope with an unfocused transducer. The transducer was positioned and angled to record the mitral anterior cusp echogram. Polaroid records of the TP scan were made from the face of the oscilloscope.

In the acute phase, ultrasonic examination was carried out with the patients in bed. At the same time, they were examined clinically with particular reference to evidence of left heart failure and mitral insufficiency. Chest x-rays, with the patients seated erect, were taken as near as was feasible to the time of the ultrasonic and clinical observations. Echocardiography was repeated in 12 patients during the acute phase of their illness.

The follow-up study was undertaken on an out-patient basis. The patients were rested for 10 minutes prior to echocardiography. All were examined clinically and 11 had chest x-rays.

The cardio-thoracic ratio was measured on the chest x-rays. Prominence of the upper lobe veins in the erect position was the minimum requirement for the radiographic diagnosis of left heart failure. For the purposes of this study, left heart failure in the acute phase, was judged to be present or absent on radiographic grounds. The radiographic and clinical assessments were in accord in 38 instances: 2 patients with doubtful clinical left ventricular failure had/..

had normal chest x-rays and were assigned to the group in which left ventricular failure was absent. At follow-up, symptoms, clinical examination and the chest x-ray when available were used to evaluate the integrity of the left ventricle.

Only mitral echograms with monophasic diastolic closure movements were selected for measurement of the diastolic closure rate (Chap. 2). More than one complex was measured in 25 acute and 14 convalescent patients, for each of whom a mean diastolic closure rate was calculated. When a patient was examined on more than one occasion in the acute phase, the diastolic closure rate recorded nearest to the time of chest x-ray was chosen for analysis.

Comparisons were made between the mitral diastolic closure rates in the acute and follow-up groups. The diastolic closure rates in both these groups were compared with those from a series of normal subjects: mean 176 mm/sec., standard deviation 29 mm/sec. (Chap. 2).

Statistical analysis was by Student's 't' tests for the significance of differences between means and between paired observations.

Results

The mitral diastolic closure rates in the acute phase are shown in Fig. 3,1 and at follow-up in Fig. 3,2. The closure rates plotted on these graphs have been expressed to the nearest 5 mm/sec. but precise values were used to calculate the means and standard deviations. The statistical significance of the differences between the diastolic closure rates in the infarct patients and normal subjects is shown in Table 3,1. Table 3,2 tabulates the significance of the differences between patient sub-groups.

I. Acute phase

All patients.

The mitral diastolic closure rate for the group was 214 $\pm$ 69 mm/sec. This was significantly higher ($P<0.005$) than normal.

Left ventricular failure present

The mitral diastolic closure rate ranged from 80 to 377 mm/sec. In 15 cases/...

cases it was above the normal range and in 4, although within the range of normal observations, it was $>$ 1SD above their mean. The diastolic closure rate was normal in 5 instances, $>$ 1SD below the normal mean in one and abnormally low (80 mm/sec) in a patient with rheumatic mitral valve disease.

The differences between the mitral diastolic closure rates of these patients and both normal subjects ($P<0.001$) and acute infarct patients without radiographic left ventricular failure ($P<0.001$), were highly significant.

Left ventricular failure absent

The mitral diastolic closure rate ranged from 123 to 213 mm/sec. While in 13 cases, the closure rate was within the range of normal observations, in one it was 1SD above their mean. An abnormally low diastolic closure rate (120 mm/sec) was recorded in one patient.

The mitral diastolic closure rates of these patients did not differ significantly from normal ($P>0.10$).

II. Follow-up

All patients

The mitral diastolic closure rate was 186 ± 63 mm/sec. This did not differ significantly from normal ($P>0.10$).

Left ventricular failure present.

The mitral diastolic closure rate ranged from 130 to 360 mm/sec. It was increased in 3 instances, within the normal range but $>$1SD above its mean in one and normal in two.

These values did not differ significantly from those observed in normals ($P<0.10$) and in convalescent infarct patients without left ventricular failure ($P<0.10$).

Left ventricular failure absent.

The mitral diastolic closure rate ranged from 83 to 275 mm/sec. While in 13 cases the closure rate was within the range of normal observations, in 2 it was $>$1SD above their mean. Increased closure rates were observed in 2 patients and low values were recorded in 3, including the patient with rheumatic mitral valve/...

valve disease.

There was no significant difference between the mitral diastolic closure rates of these patients and those of normal subjects ($P>0.10$).

Comparison of the results in the acute phase and at follow-up

The diastolic closure rates recorded from the 40 patients with acute myocardial infarction and from the 24 convalescent patients (Groups 1 and 4, Table 3,1) did not differ significantly ($P<0.10$).

24 patients were examined in both the acute and convalescent phases: the results are expressed graphically in Fig. 3,3. When Student's 't' test for paired observations was applied to these data, the differences were not significant ($P<0.10$).

Cardiac decompensation was absent in both the acute and convalescent phases in 10 patients, in 9 of whom the closure rate at follow-up was essentially unchanged or had fallen. In one patient who had developed an apical systolic murmur in the interim, the diastolic closure rate rose from 167 mm/sec to 275 mm/sec. at follow-up. The closure rates at the two examinations in this group did not differ significantly ($P>0.10$).

Left ventricular failure was present in 14 patients in the acute phase and persisted at follow-up in 6. In these 6, the diastolic closure rates at the two examinations were not significantly different ($P>0.10$) despite substantial slowing in two cases i.e. from 250 to 130 mm/sec and from 290 to 175 mm/sec.

In the 8 patients in whom left ventricular failure resolved before follow-up, the diastolic closure rate fell in 5 cases and was uncharged in two, one of whom was the patient with rheumatic mitral valve disease: the closure rate increased in only one instance. The differences between the paired observations were greatest in this group but just failed to reach statistical significance ($P<0.10$).

Papillary muscle infarction

Acute phase

A systolic murmur at or internal to the cardiac apex was heard in 9 patients with/...

with acute myocardial infarction. One was known to have rheumatic mitral valve disease. Post-mortem examination confirmed the clinical diagnosis of rupture of the inter-ventricular septum in two patients with antero-septal infarction. In one of these patients, in addition to the ventricular septal defect, there was microscopic evidence of early infarction of the base of the anterior papillary muscle. In the other, echocardiography demonstrated a pericardial effusion (Fig. 3,6): a moderate quantity of sero-sanguinous pericardial fluid was present at post-mortem.

The remaining 6 patients with murmurs survived: clinically, acute papillary muscle insufficiency was thought to be probable in 3 cases and to be almost certain in 3 others. Fig. 3,7 is the echocardiogram from a patient with classical clinical evidence of papillary muscle dysfunction.

Two patients in whom no murmur was heard were found to have papillary muscle infarction at post-mortem. One had extensive recent infarction of the free wall of the left ventricle and of both papillary muscles while the other had recent and previous posterior papillary muscle infarction.

There were thus 8 patients in whom a diagnosis of papillary muscle infarction was made at autopsy and/or on clinical examination. The diastolic closure rates for these patients have been plotted in Fig. 3,1. They are evenly distributed and do not differ significantly from the remainder of the group ($P>0.10$). When these 8 cases are excluded (Tables 3,3 and 3,4), the closure rates of acute infarcts with left ventricular failure remain significantly higher than those of normal subjects ($P<0.001$) and those of acute infarcts without failure ($P<0.001$).

Follow-up

At follow-up, apical murmurs persisted in 4 patients and had developed in 3 others. On clinical grounds, papillary muscle insufficiency was a possibility in each. The diastolic closure rates have been plotted in Fig. 3,2: they are fairly evenly distributed and do not differ significantly from the remainder of the group ($P<0.10$). Exclusion of these cases does not alter the significance of the differences between the closure rates of the patients and those/...

those of normal subjects or between the closure rates of patient sub-groups (Tables 3,3 and 3,4).

Serial measurements

Mitral echograms were recorded in 12 patients on more than one occasion during their acute illness: the diastolic closure rates have been charted (Fig. 3,4).

Patients 1 to 4 had no clinical or radiographic evidence of left heart failure. Fig. 3,5 is a representative echogram from patient 3. Patients 5 to 12 had clinical evidence of left ventricular failure at the time of each ultrasonic examination: chest x-rays to confirm the clinical assessment were not, however, taken on every occasion (Fig. 3,4). Fig. 3,7 is the acute phase echogram from patient 5 who had typical clinical signs of papillary muscle dysfunction. At follow-up the intensity of the murmur had diminished and there was no left heart failure. Patient 10 had no murmur but had posterior papillary muscle infarction at post-mortem. Patients 8, 9 and 12 also died but post-mortem examination was not carried out.

The diastolic closure rate and prognosis

The mean diastolic closure rate for the patients who died was 273 mm/sec and was significantly higher than the mean value for all other acute phase infarcts ($P<0.01$). The three fastest closure rates recorded in the series were from patients who did not survive. Further, of the 8 patients who died, 6 had diastolic closure rates above 235 mm/sec.

Discussion

The normal values against which the mitral diastolic closure rates in this study were assessed are higher than most of those published. This reflects differences in methods of recording and measurement (Chap. 2). Because care was taken with the selection of complexes for measurement, the accuracy of the diastolic closure rates for the infarct patients is acceptable, but would have been improved had a strip-chart recorder been used.

A chest x-ray taken with the patient seated erect is superior to clinical examination/...

examination in the detection of early left heart failure complicating acute myocardial infarction (Chait et al., 1972). In the acute phase of this investigation, cardiac decompensation was therefore diagnosed on the basis of the radiographic findings.

Killip and his co-workers have found the left ventricular end-diastolic pressure to be consistently elevated when left heart failure, even of mild degree, complicates acute myocardial infarction (Wolk et al., 1972; Scheidt et al., 1973). Their criteria for the recognition of mild left heart failure, "rales thought to be of cardiac origin over an area of less than half of both lung fields", were less rigorous than those adopted in the present study. Thus, it is reasonable to assume that the left ventricular end-diastolic pressure was elevated in the patients judged here to be in left heart failure.

The results indicate that whereas the diastolic closure rate of the mitral valve is normal in uncomplicated acute myocardial infarction, it is increased in acute myocardial infarction complicated by left heart failure. There is also evidence to suggest that the diastolic closure rate returns to normal with resolution of left heart failure: however, the number of observations was small and the findings were not statistically significant. Further, the mitral diastolic closure rate has been found to be normal in convalescent infarct patients without heart failure.

It is clear from these observations that the diastolic closure rate of the normal mitral valve increases in the presence of radiographic evidence of left heart failure. It is therefore concluded that in the context of acute myocardial infarction, the left ventricular filling pressure is a major determinant of the mitral diastolic closure rate. However, a few patients had normal diastolic closure rates despite the presence of left heart failure. These exceptions suggest that the diastolic closure rate has additional determinants.

Layton et al. (1973) reached similar conclusions. They were, however, unable to distinguish between patients with high and those with normal left ventricular end-diastolic pressures on the basis of the diastolic closure rate and produced some evidence that ventricular diastolic compliance is a further factor determining the/...

the mitral diastolic closure rate. The highest diastolic closure rate they observed was 170 mm/sec: reduced ventricular compliance may well have been responsible for the slow closure rates as most of their patients had established left ventricular disease. Nonetheless, the diastolic closure rates reported by these workers in normal subjects are so widely at variance with those observed by others, that the validity of their measurements can reasonably be doubted (Chap. 2).

It is likely that the diastolic closure rate has multiple determinants. Thus, in a model left ventricle, mitral valve flow and vortex formation within the ventricle have been shown to play a part in valve closure (Bellhouse, 1972). Further, if the diastolic closure rate reflects the rate of left ventricular filling (Layton et al., 1973), then left atrial and left ventricular size, performance and compliance and all the factors influencing them may be involved.

Left ventricular compliance may be reduced in acute myocardial infarction (Diamond and Forrester, 1972). When the present study was undertaken it was anticipated that this abnormality would be reflected in slow diastolic closure rates. This has proved not to be so; with the exception of the patient with rheumatic heart disease, no abnormally low values were recorded. Reduced ventricular compliance could, however, have accounted for the normal closure rates found in the presence of left heart failure in 4 acute phase and 2 convalescent patients. Reduced ventricular compliance was thought to be likely in two of these patients with acute myocardial infarction. Thus, one of them had a persistent sinus tachycardia and a substantial pericardial effusion while the other, who pursued an intractable course with a sinus tachycardia and gross pulmonary oedema was found at post-mortem to have almost total left ventricular free wall infarction. The other two patients had electrocardiographically localised infarcts and responded readily to anti-failure therapy: an important reduction in ventricular compliance was considered to be unlikely.

Tallury, De Pasquale and Burch (1972) found widely fluctuating diastolic closure rates in infarct patients with auscultatory evidence of papillary muscle dysfunction: the results in patient 5 (Fig. 3,4) conform to the pattern they observed.

Echocardiographic/...

Echocardiographic features diagnostic of papillary muscle dysfunction have not, however, been reported. The problems of clinical diagnosis have been highlighted here by the two cases of extensive papillary muscle infarction detected only at post-mortem. The contribution of ischaemic mitral regurgitation to the high diastolic closure rates found in patients with left heart failure must thus remain uncertain. It is, however, important to note that exclusion of patients with probable or proven papillary muscle dysfunction from the analysis did not alter the general conclusions.

Looking at the results in practical terms, there is evidence that measurement of the diastolic closure rate in acute myocardial infarction may not only differentiate between patients with normal and those with high left ventricular pressures but may, when considered in conjunction with the radiographic findings, identify patients in whom left ventricular compliance is reduced. It is likely that further useful information could be obtained if the diastolic closure rate were considered in the light of simultaneous ultrasonic assessments of left ventricular dimensions and performance.

Summary

Cardiac lesions in which the diastolic closure rate of the mitral valve is increased and others in which it is decreased have been defined. These established associations suggest that left ventricular filling and the mitral diastolic closure rate are related.

To investigate its relation to left ventricular filling pressure, the mitral diastolic closure rate was measured in the acute (40 patients) and again in the convalescent (24 patients) phase of myocardial infarction. Left ventricular filling pressure was judged to be normal or elevated on radiographic grounds.

In the acute phase, the diastolic closure rate was increased in the presence of radiological left heart failure ($P<0.001$) and was normal in uncomplicated myocardial infarction. In the convalescent phase, the diastolic closure rate was normal in patients with ($P<0.10$) and in those without ($P>0.10$) left/...

left heart failure. When left heart failure resolved the diastolic closure rate fell, but not significantly ($P>0.10$).

It is concluded that, in the context of acute myocardial infarction, left ventricular filling pressure is a major determinant of the mitral diastolic closure rate. It is suggested that a reduction in left ventricular compliance should be suspected when radiographic left heart failure complicating myocardial infarction is associated with a normal mitral diastolic closure rate.

TABLE 3,1 ALL PATIENTS

	n	DCR mm./sec. Mean	SD	Difference from normals P
Normals	45	176	29	
A. Acute phase infarcts				
1. All patients	40	214	69	< 0·005
2. LVF present	26	241	70	< 0·001
3. LVF absent	14	165	28	> 0·10
B. Follow-up infarcts				
4. All patients	24	186	63	> 0·10
5. LVF present	6	234	81	< 0·10
6. LVF absent	18	170	48	> 0·10
Combining A and B				
7. All patients	64	204	67	< 0·005
8. LVF present	32	240	71	< 0·001
9. LVF absent	32	168	40	> 0·10

TABLE 3,2
ALL PATIENTS

GROUPS	P
1 & 4	> 0·10
2 & 3	< 0·001
5 & 6	< 0·10
8 & 9	< 0·001

TABLE 3,3

PATIENTS WITHOUT PAPILLARY MUSCLE DYSFUNCTION

	n	DCR mm/sec. Mean	SD	Difference from normals P
Normals	45	176	29	
A. Acute phase infarcts				
1. All patients	32	209	67	< 0·02
2. LVF present	20	236	69	< 0·001
3. LVF absent	12	163	28	> 0·10
B. Follow-up infarcts				
4. All patients	17	174	66	> 0·10
5. LVF present	3	249	115	> 0·10
6. LVF absent	14	157	42	> 0·10
Combining A and B				
7. All patients	49	197	68	< 0·10
8. LVF present	23	238	73	< 0·001
9. LVF absent	26	160	35	< 0·10

TABLE 3,4

PATIENTS WITHOUT PAPILLARY MUSCLE DYSFUNCTION

GROUPS	P
1 & 4	< 0·10
2 & 3	< 0·001
5 & 6	> 0·10
8 & 9	< 0·001

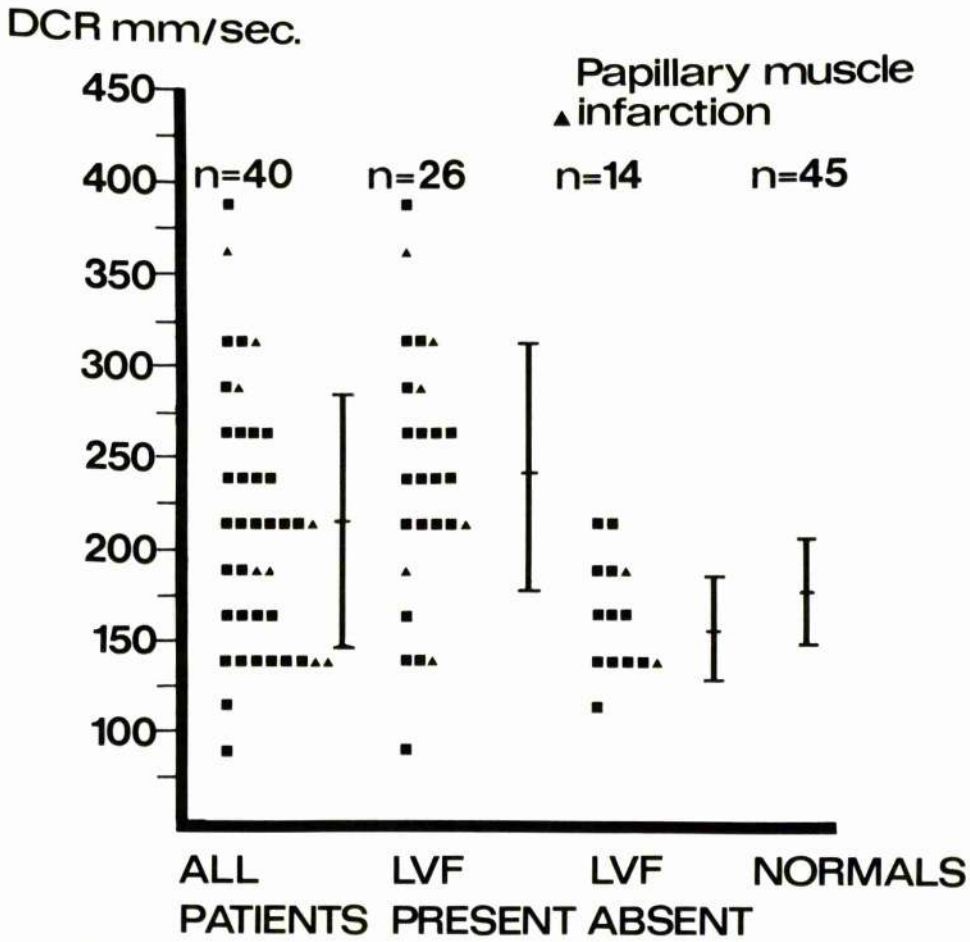


FIG. 3,1 ACUTE PHASE

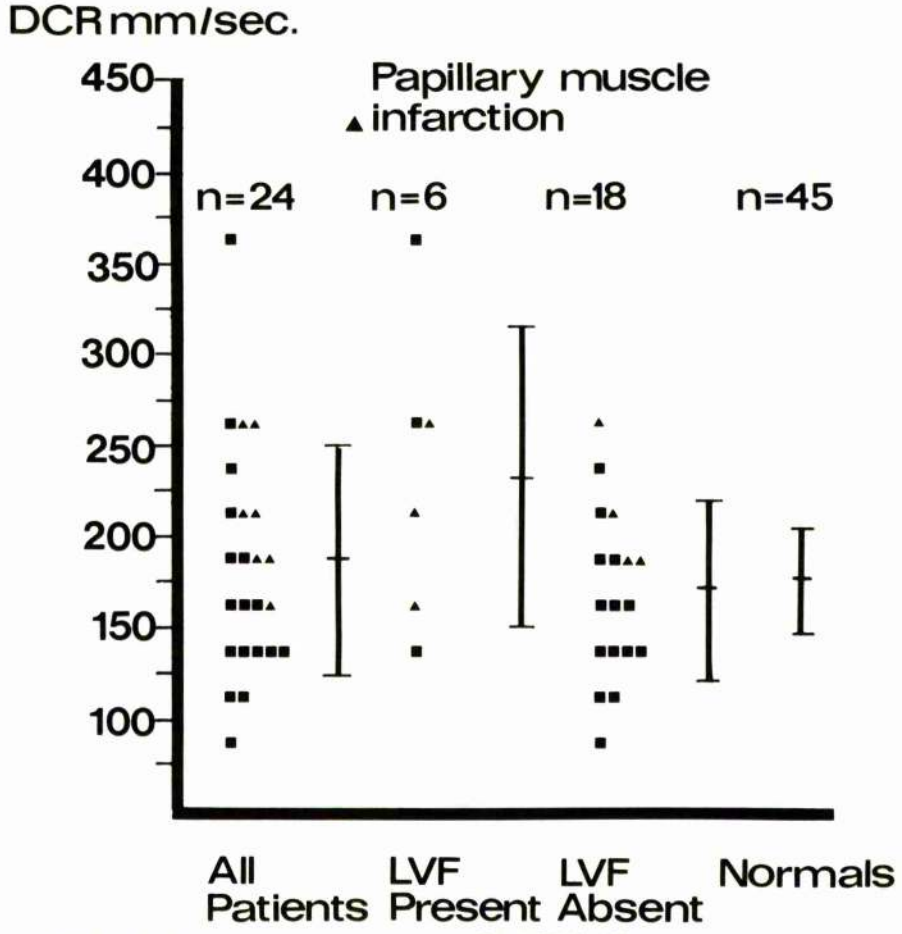


FIG. 3,2 FOLLOW-UP

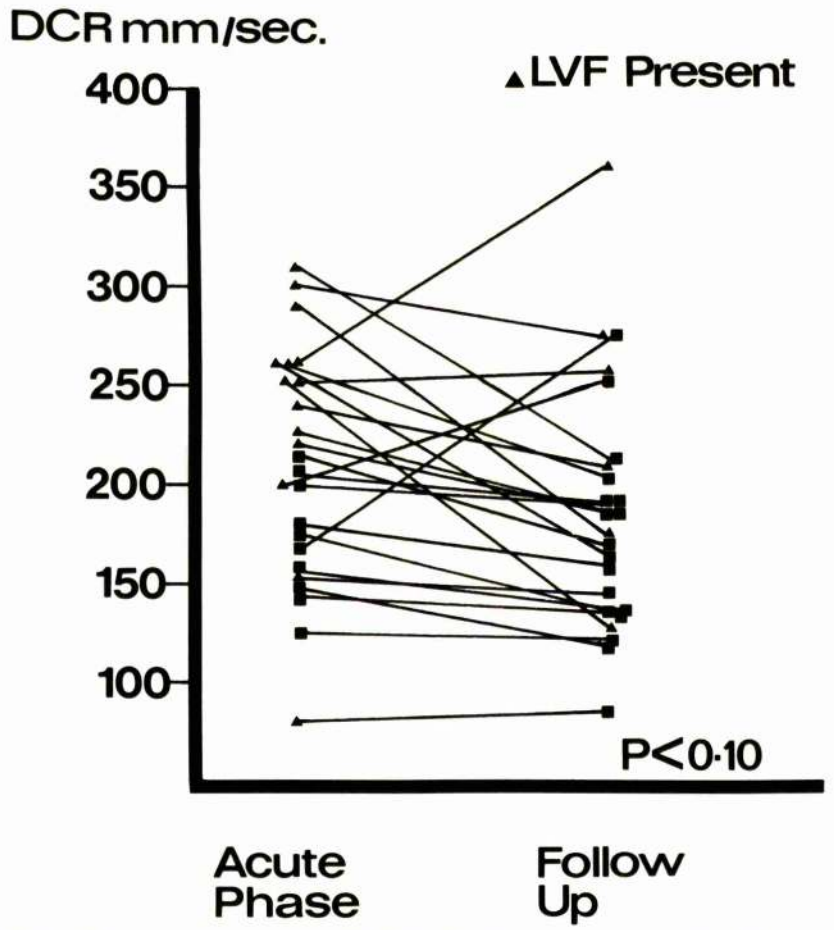


FIG. 3,3 DIASTOLIC CLOSURE RATE IN THE ACUTE AND CONVALESCENT PHASES - 24 PATIENTS

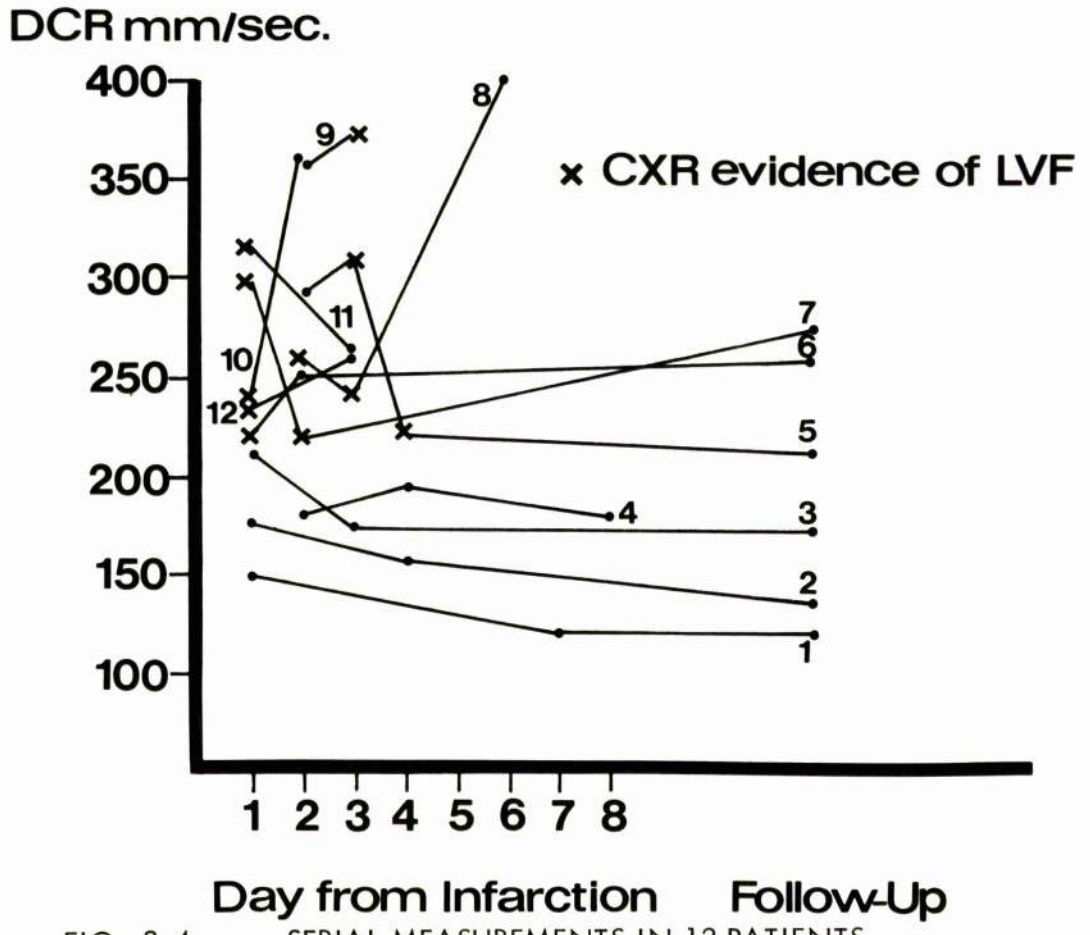


FIG. 3,4 SERIAL MEASUREMENTS IN 12 PATIENTS

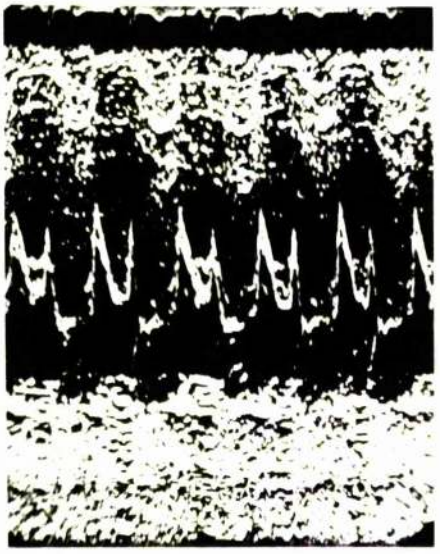


FIG. 3,5 CASE 3, FIG. 3,4

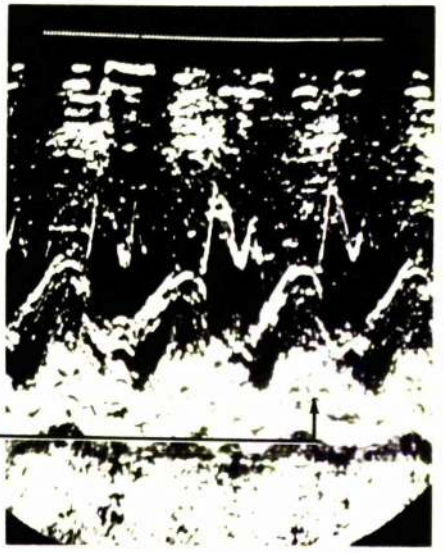


FIG. 3,6 PERICARDIAL EFFUSION COMPLICATING MYOCARDIAL INFARCTION

FIG. 3,7 CASE 5, FIG. 3,4

CHAPTER 4

ECHOCARDIOGRAPHY OF THE LEFT HEART

If misinterpretation of echocardiographic information is to be avoided, it is essential to have a detailed knowledge of the origins and the inter-relations of the echoes which can be recorded from the normal and the abnormal heart.

Edler made a basic contribution when, from anatomical considerations and from studies on the isolated animal heart, he identified the mitral anterior cusp echogram and thus established a point of reference from which the origins of other echoes could be deduced (Edler, 1961; Edler et al., 1961). Subsequently, the echoes from several major intra-cardiac structures were identified and inter-related by ultrasonic contrast studies (Gramiak, Shah and Kramer, 1969) and more recently, strip-chart recordings have been used to display these inter-relations (Feigenbaum, 1972). However, the echocardiographic features of some intra-cardiac structures have not been fully described and there is evidence in the literature that, possibly as a consequence, echocardiographic findings have occasionally been misconstrued.

In this account, records from normal subjects and from patients with non-rheumatic heart disease are used to illustrate in detail the echocardiographic features of the left heart.

Material

Ultrasonic records from normal subjects and from patients with non-rheumatic heart disease were reviewed. Those which displayed the inter-relations of left heart structures and which were technically satisfactory were selected and form the basis of this account.

Methods

The equipment used was an Eskoline 20 ultrasonoscope with an unfocused transducer. Polaroid or strip-chart (Honeywell Visicorder) records were made : on these, tissue depth and time markers were recorded as previously described (Chap 2). With the patients supine or seated erect, the position and angulation of/...

of the transducer were altered freely to display the intra-cardiac structures.

Discussion

The anatomy of the normal left heart is illustrated diagramatically in Fig. 4,1. The paths of the ultrasonic beam which have been superimposed are referred to subsequently.

Aortic root

Gramiak and his co-workers described the aortic root echogram and showed that it is related posteriorly to the left atrium (Gramiak et al., 1969). They later used the depth of the left atrial cavity behind the aortic root at end-systole as a measurement of left atrial size (Gramiak and Shah, 1971). This measurement cannot be precise because there are no points of reference from which the path of the ultrasonic beam in the left atrium can be deduced. Thus, in recording the aortic root echograms in Figs. 4,2 to 4,4, the beam may have been directed to traverse the left atrium obliquely as in Fig. 4,1, path 1 or it may have been directed more superiorly to traverse the short axis of the chamber, immediately behind the aortic root : the apparent left atrial size will differ in the two circumstances.

Systolic flutter of the aortic leaflets is an abnormal but non-specific finding : Figs. 4,2 and 4,3 were recorded from a patient with left heart failure, presumed to be on a hypertensive basis.

The aortic root is in continuity below with the interventricular septum and with the mitral ring and the root of the mitral anterior cusp (Figs. 4,3 and 4,4). Echoes from the aortic root and the immediately subjacent portion of the mitral anterior cusp are recorded at the same depth (Johnson et al., 1972). As the ultrasonic beam is angled towards the free edge of the anterior cusp and downwards towards the left ventricle, the aortic root and the anterior leaflet no longer lie in the same plane (Fig. 4,1). Systolic prolapse of the anterior leaflet can thus only be diagnosed from the mitral echograms in the first part of a continuous downwards sweep from the aortic root.

Left atrial wall

In Fig. 4,4 the posterior wall of the left atrium is recorded as a pulsatile structure/

structure behind the mitral anterior cusp : it moves posteriorly in ventricular systole and during ventricular diastole exhibits two anterior movements, the second of which co-incides with atrial systolic re-opening of the anterior cusp. Although the aortic root is also related posteriorly to the left atrium, a clear waveform from the wall of that structure has not been recorded behind the aortic root echogram, suggesting that the ultrasonic beam has cut the atrial wall obliquely, possibly as indicated in Fig. 4,1, path 1.

Fig. 4,5 shows the transition from a left atrial wall to a left ventricular wall echogram as the beam is directed through the atrio-ventricular junction (Fig. 4,1, path 3) towards the left ventricle.

I have found no detailed description of the left atrial echogram in the literature. However, Johnson et al. (1972) and Feigenbaum (1973) have published tracings which include unlabelled left atrial wall records.

Mitral ring

The mitral orifice is surrounded by a fibrous ring which is continuous on its inner aspect with the valve leaflets. Angiographic studies have shown that the mitral ring moves downwards and forwards towards the cardiac apex during ventricular systole and upwards and backwards towards the base of the heart in diastole (Dayem et al., 1967). In Fig. 4,1, its end-diastolic and end-systolic positions have been represented diagramatically.

The mitral ring and the portion of the left ventricular wall below the free margins of the cusps (Fig. 4,1, path 4) move in directions which are essentially at right angles; in systole the ring moves downwards and forwards along the axis joining the mitral orifice and the apex while the left ventricular wall moves upwards and forwards across the mean minor axis of the ventricle (Gibson and Brown, 1973). It can be concluded that echoes within the left ventricle moving in parallel with its posterior wall cannot originate from the ring and that mitral ring echoes will be displayed only when the ultrasonic beam is directed towards the left atrium. Further, because the ring and the leaflets are in continuity, mitral ring echoes will be recorded when the beam is directed towards the roots of the cusps.

In/...

In Fig. 4,6, adjacent to the typical anterior cusp echogram, there is a stronger echo : the two are variably related. Thus, in ventricular diastole the stronger echo parallels the closure movement of the anterior cusp, intervenes between it and the left atrial wall and can be seen to be in continuity with it at the onset of atrial systole. During ventricular systole both echoes move anteriorly : throughout most of this phase the anterior cusp echogram is recorded behind the stronger echo, intervening between it and the left atrial wall. In early diastole as the anterior cusp moves rapidly forward to its open position, it is briefly obscured by the stronger echo.

In the normal heart, the free edges of the two mitral leaflets move in opposite directions during diastole : there is no anatomical or haemodynamic evidence which suggests that the direction of movement of the root of the posterior cusp differs from that of its free margin or that the root of the posterior cusp is in apposition with the anterior cusp during this phase of the cardiac cycle. The strong echo moving with the anterior cusp in diastole and in apparent continuity with it in pre-systole (Fig. 4,6) cannot therefore be derived from the posterior cusp and must originate from the mitral ring.

In the normal heart during systole, the anterior leaflet does not prolapse behind the posterior one but both are known to bulge towards the left atrium (Burch, De Pasquale and Phillips, 1963). In Fig. 4,6, the anterior cusp is displayed within the left atrium behind the stronger echo, providing further evidence that this echo is derived from the mitral ring.

It is suggested that a complete echogram from the mitral ring can be recorded only when the ultrasonic beam is directed, as in Fig. 4,1, path 2, along its line of motion. While it is appreciated that Fig. 4,1, path 2 does not precisely represent the course of the beam in Fig. 4,6, it does explain the sequence in which the echoes have been recorded. Thus, in both Figs. the beam consecutively traverses the septum, the anterior cusp, the ring and the left atrial wall in diastole and the septum, the ring, the anterior cusp and the left atrial wall in systole.

In my experience it is difficult to record a continuous echogram from the mitral/...

mitral ring. However, strong echoes which move anteriorly in systole and posteriorly in diastole are frequently seen adjacent to the anterior cusp when it is displayed with the left atrium behind it (Figs. 4,5; 4,7; 4,8 and 4,9). Theoretically, these echoes are likely to be from the ring : failure of the ultrasonic beam to traverse that structure at right angles would account for their distorted waveform. Thus, in Fig. 4,8, echoes thought to be from the ring have been recorded but the beam has traversed the left atrium close to its junction with the ventricle and could not have been directed along the line of movement of the ring.

Because the echograms from the mitral ring, the chordae tendinae and the left ventricular posterior wall are similar, there has undoubtedly been confusion in their identification. For example, inspection of the tracings in the original account of the mitral ring echogram (Zaky, Grabhorn and Feigenbaum, 1967) suggests that the echoes described originated in fact from the endocardium of the left ventricle and from the chordae. Further, Chakorn et al. (1972) misinterpreted echoes from the left ventricular endocardium and from the rheumatic mitral posterior cusp.

Ring movement undoubtedly contributes to the mitral diastolic closure movement (Fig. 4,6). It is, however, difficult to record accurately and the practicality of proposals to incorporate measurements of the amplitude of ring movement in the ultrasonic assessment of left ventricular filling (Layton et al., 1973) is doubtful.

Left ventricular wall

The left ventricular wall echogram alters as the ultrasonic beam is swept from the atrio-ventricular junction downwards through the left ventricle of a normal heart. Figs. 4,9 and 4,10 are from the same subject : the thickness of the left ventricular wall increases with downward angulation of the transducer and its excursion is 8 mm and 14 mm in the first and last parts of the sweep respectively. It is contended that these differences are real and not technical and that they occur as the beam is directed on to the posterior papillary muscle. Thus, when left ventricular wall excursion and thickness are maximal, the posterior/...

posterior cusp echogram is replaced by echoes from the chordae tendineae (Figs. 4,9 and 4,10) suggesting that the beam has passed through the free margin of the anterior cusp and beyond the free margin of the shorter posterior one, to the region of the posterior papillary muscle (Fig. 4,1, path 5). Conversely, when the beam traverses the free margin of the posterior cusp (Figs. 4,9; 4,10 and Fig. 4,1, path 4) to which the posterior papillary muscle cannot be subjacent, left ventricular wall excursion and thickness are submaximal. These observations are represented diagrammatically in Fig. 4,11. It is of practical importance to note that end-systolic dimension is smaller at the papillary muscle than at the level of the free margins of the cusps and that the derived stroke volume is therefore different at the two sites.

Posterior cusp and chordae tendineae

Fig. 4,5 was recorded as the beam was directed from the left atrium to the left ventricle : at end-diastole a thin echo moves rapidly from the atrial wall towards the anterior cusp. Similar echoes are present in Figs. 4,7 to 4,10 and 4,12 : there is an apparent transition to a typical posterior cusp free margin echogram in Fig. 4,10. In Figs. 4,5 and 4,10 the beam must have been directed much as in Fig. 4,1, path 3. The thin echo is thus likely to be from the root of the posterior cusp : this part of the cusp moves into the atrium in systole as its free margin closes against the anterior cusp within the ventricle.

As indicated previously, echoes from the posterior cusp cease to be recorded when the transducer is angled downwards from its free margin into the ventricle (Figs. 4,9 and 4,10). They are replaced by stronger echoes which move anteriorly in systole and posteriorly in diastole but which lack the diastolic closure and pre-systolic re-opening movements characteristic of the leaflet echogram (Fig. 4,13). As the beam sweeps on downwards through the ventricle, similar echoes also replace the anterior cusp echogram (Fig. 4,14) : it is concluded that they originate from the chordae tendineae.

Interventricular septum

In Fig. 4,5, the echoes from the interventricular septum alter as the beam sweeps downwards from the atrium through the ventricle. The septum appears to become/...

become thicker with downward angulation of the transducer and the amplitude of septal motion increases from 4mm in the first to 8mm in the last part of the sweep. These changes may simply be technical, resulting from alteration in the angle at which the beam traverses the septum. They may, however, be real and may reflect the transition between the membranous and the thicker muscular portions of the interventricular septum. This conclusion is obviously highly speculative but tends to be supported by the fact that the change in waveform is abrupt and is consistently recorded as the beam passes through the atrio-ventricular junction. Thus, in Fig. 4,10 the septal echogram alters after the second complex, when the beam passes to the left ventricle and is unchanged thereafter despite a downward sweep to the region of the posterior papillary muscle. A similar sequence is seen in Fig. 4,8. It is also present, although less obviously, in Fig. 4,9 where transition of the septal waveform commences at the point where a distorted left ventricular wall echogram is first recorded. The aortic root is continuous anteriorly and below with the membranous portion of the septum (Fig. 4,1). In Fig. 4,15, the thinner less pulsatile septal echogram has been recorded below the aortic root, favouring its origin from the membranous septum.

These observations cannot be further validated at present. It is interesting to note that although the echoes from the interventricular septum have been studied by a number of workers (Popp et al., 1969; Pombo, Troy and Russell, 1971; Diamond et al., 1971; Ludbrook et al., 1973), none has commented on the two distinct septal echograms reported here.

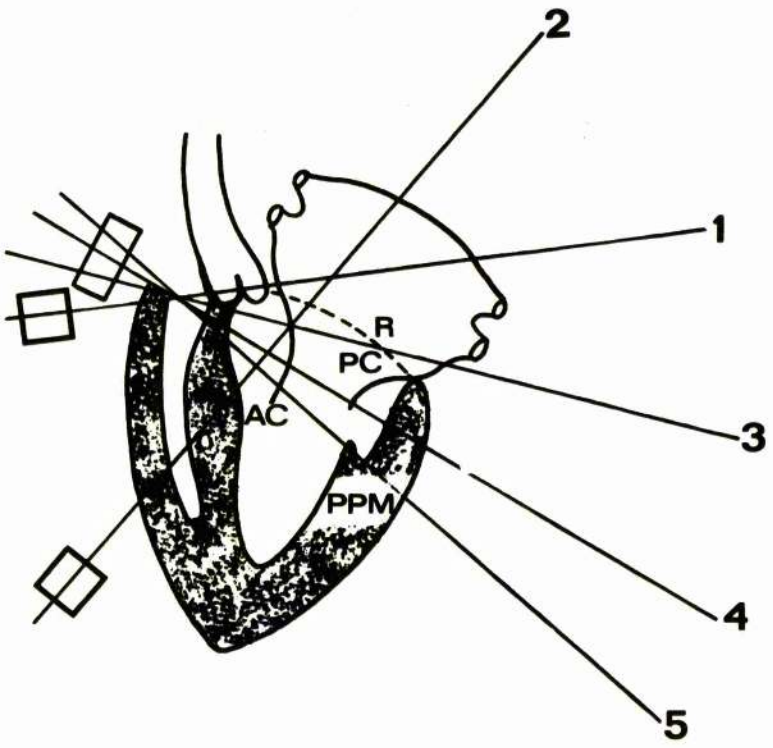
This account has dealt comprehensively with the echocardiographic anatomy of the left heart. It includes new observations which I consider to be valid and which have been substantiated asfar as a single dimensional approach will allow. The multidimensional system developed by Bom and his co-workers (Kloster et al., 1973; Roelandt et al., 1974) is an important advance: using it, it should be possible to obtain additional information on the inter-relations of cardiac echoes.

Summary

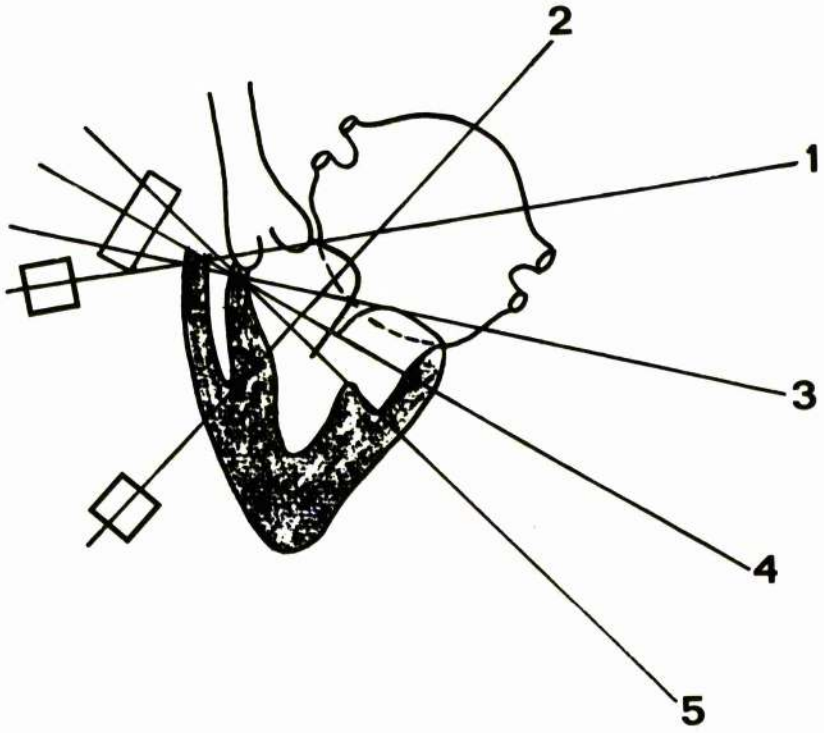
To/...

To investigate the echocardiographic features of the normal left heart, echograms were recorded as the angulation of the ultrasonic beam was altered and with a fixed beam. The Polaroid and strip chart records obtained are presented: they display left heart echoes and their inter-relations.

It is concluded: 1) Echoes from the mitral ring are never recorded within the left ventricle. 2) The left atrial wall echogram has a characteristic waveform. 3) Echoes from the root of the posterior cusp can be recorded within the left atrium. 4) The waveform of the posterior cusp echogram alters as the ultrasonic beam is swept from the root to the free margin of the cusp. 5) In the region of the posterior papillary muscle, the posterior cusp echogram is replaced by echoes from the chordae tendineae which have a characteristic waveform. 6) Two distinct echocardiographic patterns can be recorded from the interventricular septum: it is suggested that these originate from its membranous and muscular portions.



DIASTOLE



SYSTOLE

FIG. 4,1 THE NORMAL LEFT HEART

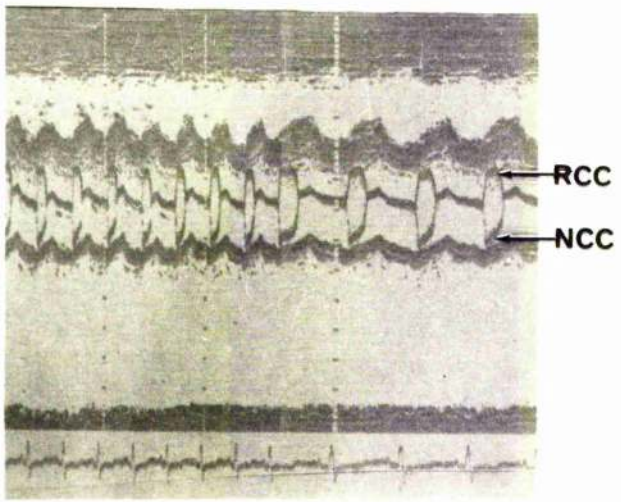


FIG. 4,2 FOR THIS AND SUBSEQUENT FIGS. - See Text

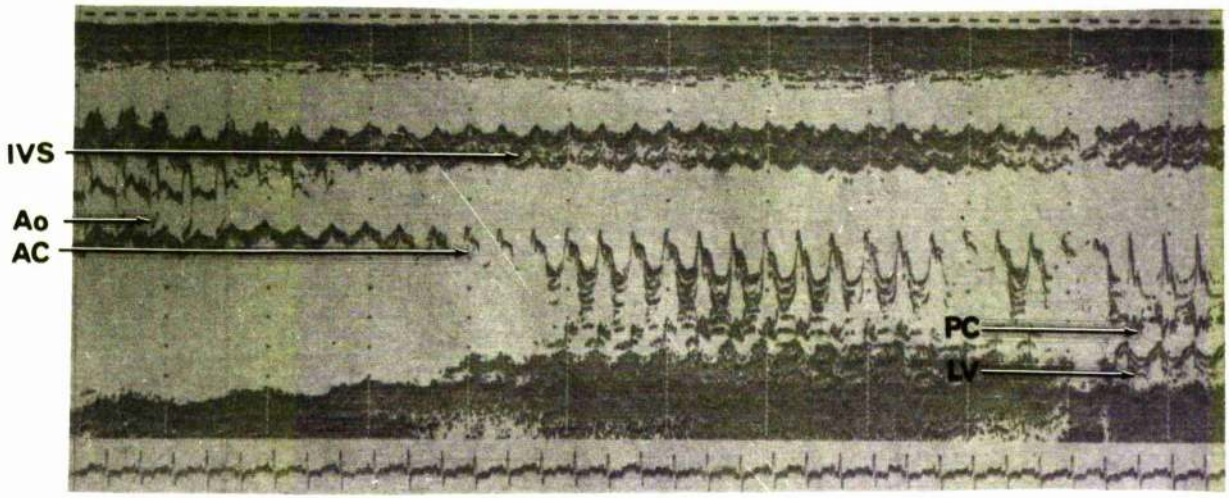


FIG. 4,3

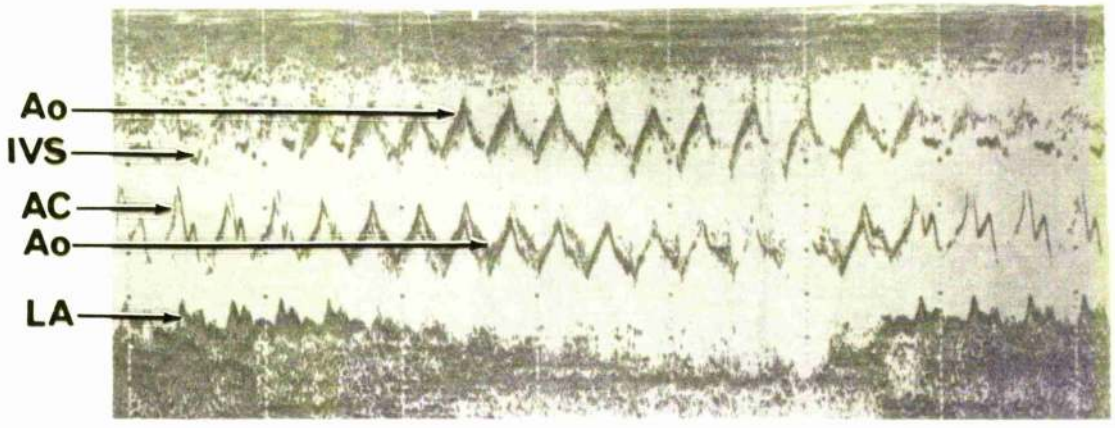


FIG. 4,4

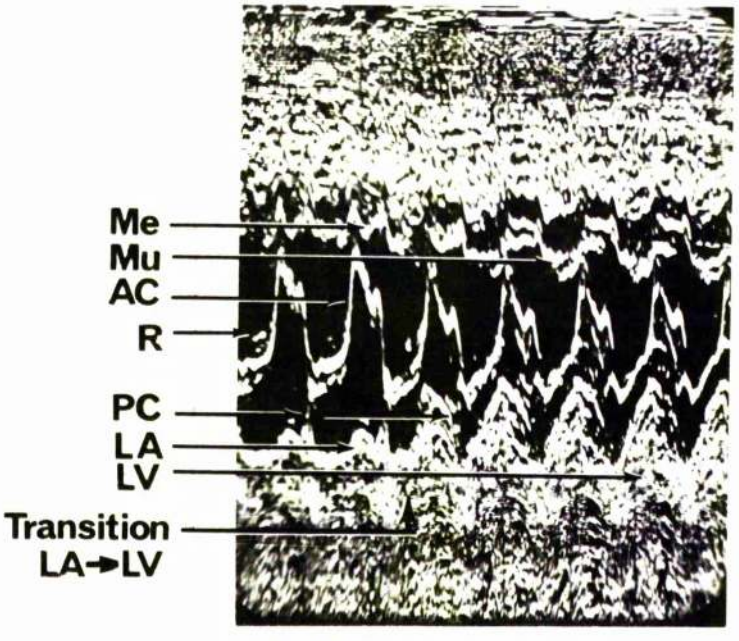
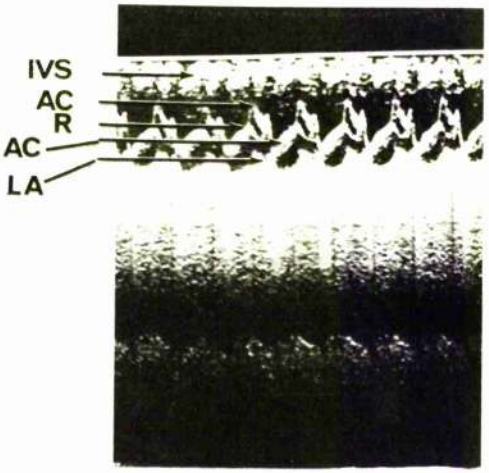


FIG. 4,5



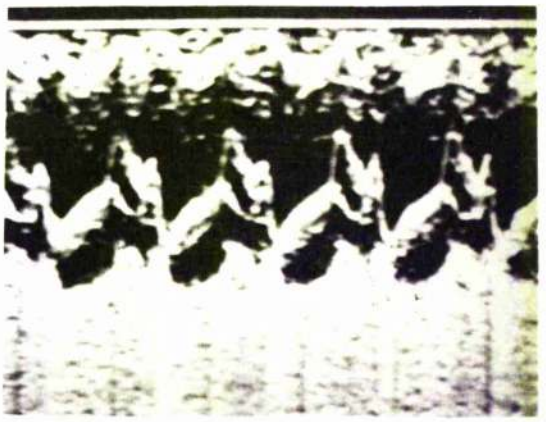


FIG. 4,6

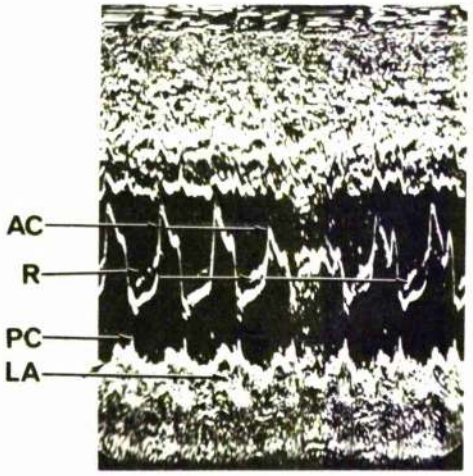


FIG. 4,7

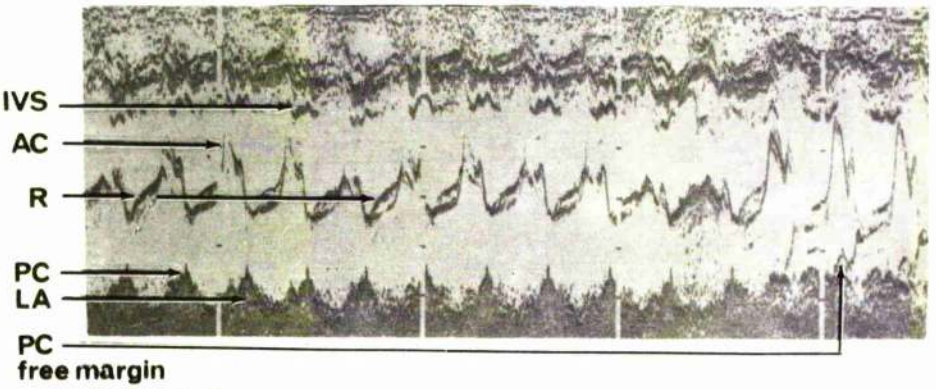


FIG. 4,8

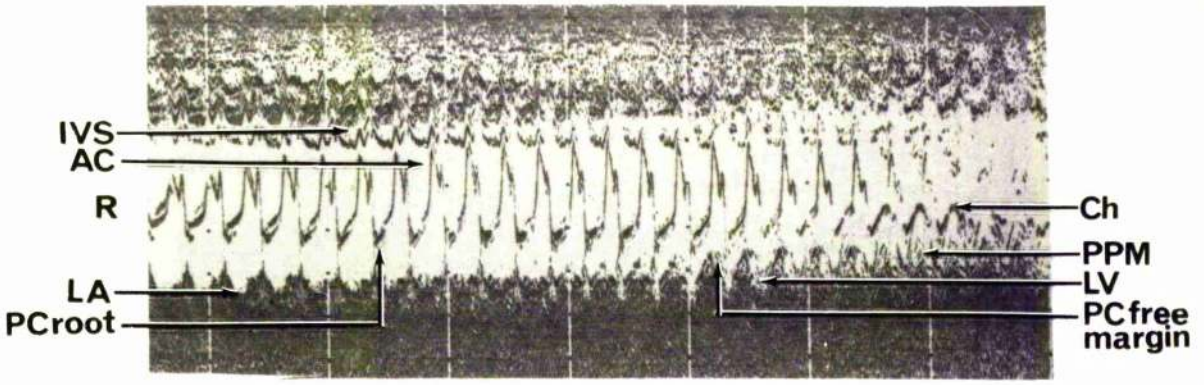


FIG. 4,9

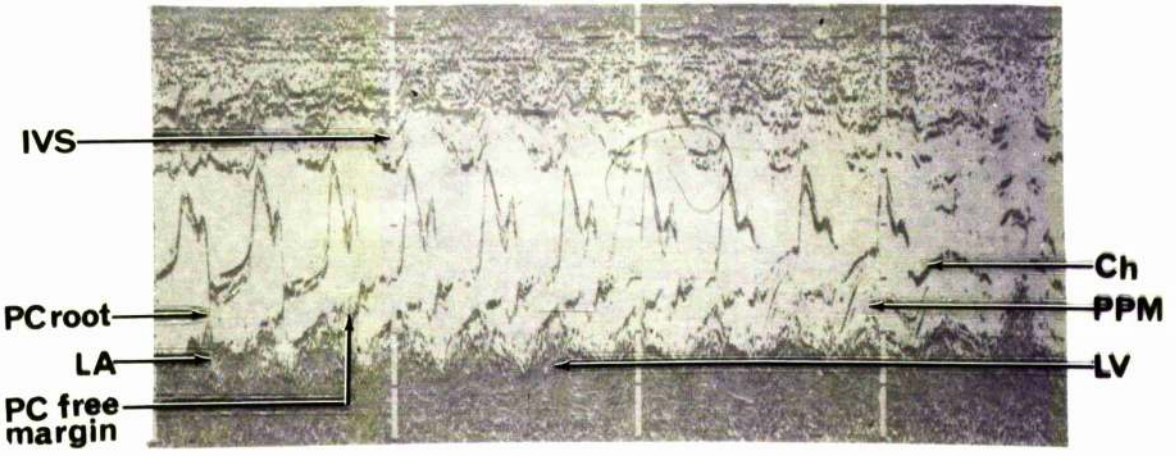


FIG. 4,10

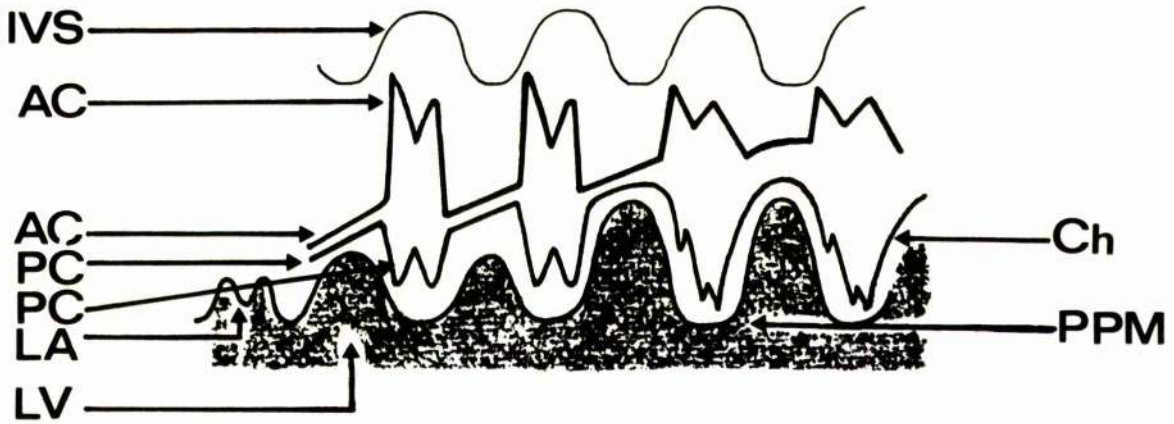


FIG . 4,11

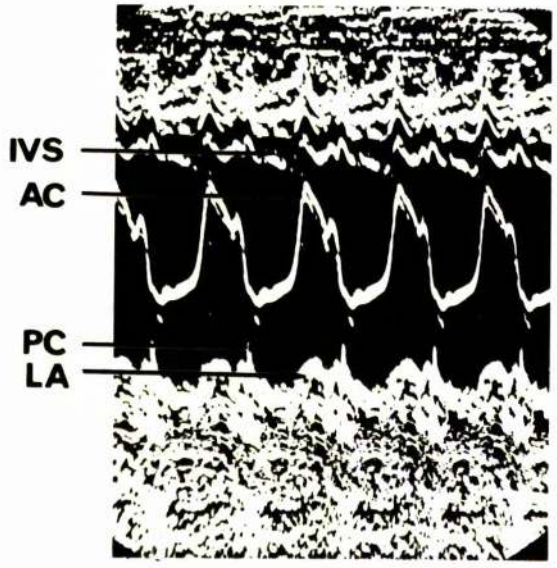


FIG . 4,12

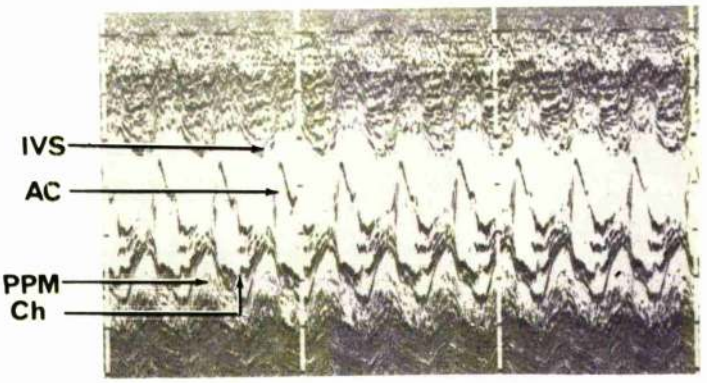


FIG. 4,13

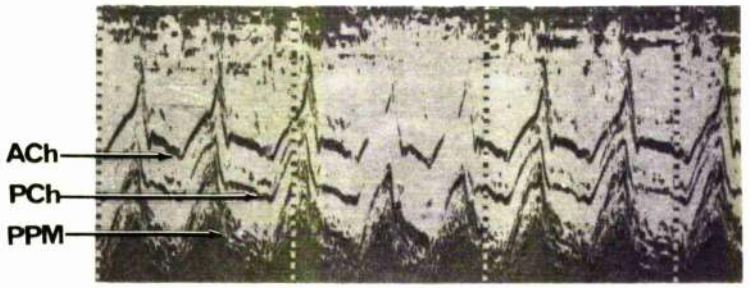


FIG. 4,14

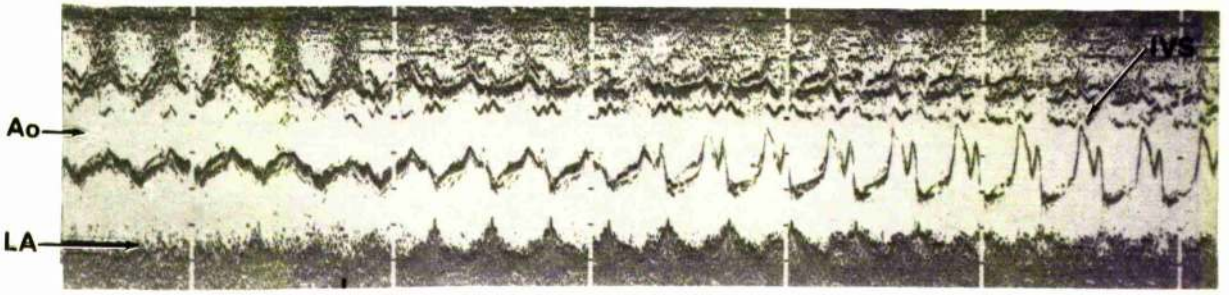


FIG. 4,15

CHAPTER 5

THE RHEUMATIC MITRAL VALVE

Following the identification of the mitral anterior cusp echogram (Edler, 1961; Edler et al., 1961), a number of workers reported on the use of ultrasound in the investigation of the rheumatic mitral valve (Effert et al., 1964; Segal, Likoff and Kingsley, 1966; Edler, 1967; Wharton and Lopez Bescos, 1970). They showed that echocardiography can provide useful information on the structure and function of the rheumatic valve and it is evident from the subsequent literature that their studies are widely regarded as definitive. However, it is worth noting that, with the exception of Segal and his co-workers, these investigators used systems which recorded single echoes and they were thus not in a position to comment either on ultrasonic evidence of calcification or thickening of the anterior leaflet or on echoes from other parts of the mitral valve apparatus. For these reasons and because the accuracy of measurements made from records other than strip-chart records is questionable (Chaps. 1 and 2) I found it necessary, when setting up a routine service in cardiac ultrasound, to re-evaluate even this best accepted application of the method.

Patients

Two groups were studied.

Group 1

24 patients considered on haemodynamic and/or clinical evidence to be unequivocally suitable for closed mitral valvotomy. All underwent closed valvotomy (Mr. I.A. Sallam). All had auscultatory evidence of mitral stenosis which was judged clinically to be severe in 12, moderate in 10 and mild in 2. A trivial aortic valve lesion was present in 2 cases. Pre-operative right heart catheterisation was performed in 13 patients and 2 had an additional left heart study. Fluoroscopy was carried out only in the patients who were catheterised: it demonstrated mitral valve calcification in one case.

Mitral/...

Mitral regurgitation of insufficient severity to preclude closed valvotomy was suspected clinically in 8 cases: minor mitral regurgitation was confirmed in the 2 submitted to angiography.

Group 2

13 patients whose suitability for closed valvotomy was doubted on clinical and haemodynamic grounds. Three underwent open valvotomy and 10 had a closed procedure: the operations were performed by 6 different surgeons. All patients had auscultatory evidence of mitral stenosis which was judged clinically to be severe in each. A haemodynamically significant aortic valve lesion was present in 3 cases. Pre-operative right heart catheterisation was performed in all patients and 6 had an additional left heart study. Fluoroscopy was carried out in all cases and demonstrated mitral valve calcification in 4 of them.

Minor mitral regurgitation was suspected clinically and confirmed angiographically in 6 cases.

Methods

Echocardiograms were recorded in all 37 patients before operation and the examination was repeated in 35 patients on one occasion between the second and eighth post-operative weeks: 2 patients from Group 1 were lost to follow-up.

For the purposes of this study, the surgeon who performed the valvotomies in Group 1 made detailed notes of his operative findings and procedure. The information in Group 2 was less precise and was extracted from the surgeons' routine operation notes.

The ultrasound findings were compared with the results of cardiac catheterisation, the operative findings and the clinical assessment of the mitral valve.

An Eskoline 20 ultrasonoscope and an unfocused transducer were used in this study. The patients were examined supine and the transducer was positioned and angled to record the mitral anterior cusp echogram. Records were made with a strip-chart recorder (Honeywell Visicorder) at chart speeds of 10 and 20 mm/sec or/...

or the TP display was photographed directly from the oscilloscope face.

Fig. 5,1 shows how the amplitude of the mitral echogram was measured from the records and how the diastolic closure rate was measured when the diastolic closure movement had a single component i.e. was monophasic. When the diastolic closure movement had two components i.e. was biphasic, the method of measurement of the diastolic closure rate was less conventional. Thus, when as in complex (a), Fig. 5,2, the duration of the first component of the diastolic closure movement was at least 40% of its total duration, the diastolic closure rate was measured from the first component and the second was disregarded. When the duration of the first component of the diastolic closure movement was less than 40% of its total, the mean diastolic closure rate was measured as in complex (b), Fig. 5,2.

Multiple echoes behind and parallel to the main cusp echo (Fig. 5,3) were taken as evidence of mitral calcification, provided that they persisted when the intensity of the ultrasonic beam was reduced to the minimum necessary to record a continuous anterior cusp echogram.

Results

Amplitude of the mitral echogram

The amplitude of the echograms recorded in Groups 1 and 2 are detailed in Figs. 5,4 and 5,5 respectively. They are compared with the amplitudes (22 - 41 mm) recorded in a series of normal subjects.

Before operation, abnormally low amplitude echograms were recorded in 5 patients from Group 1 and in 5 from Group 2. The mean pre-operative amplitude in the smaller Group 2 was below the normal range. Echogram amplitude increased, but not greatly, with valvotomy. Thus, the mean amplitude rose from 26.0 ± 4.4 mm to 27.3 ± 4.5 mm in Group 1 and from 21.7 ± 4.4 mm to 22.4 ± 4.0 mm in Group 2. Post-operatively, the amplitude remained abnormally low in 2 patients from Group 1 and in 4 from Group 2.

Mitral diastolic closure rate

The/...

The mitral diastolic closure rates recorded in Groups 1 and 2 are detailed in Figs. 5,6 and 5,7 respectively. The pre-operative values were low, being less than 25 mm/sec. in 29 cases. The diastolic closure rate increased with valvotomy. Thus, the mean value rose from 18.8 ± 13.1 mm/sec. to 49.4 ± 11.6 mm/sec. in Group 1 and from 11.4 ± 8.4 mm/sec. to 29.7 ± 15.2 mm/sec. in Group 2. The highest diastolic closure rate recorded after valvotomy was 70 mm/sec. All the post-operative closure rates were therefore well below the normal range observed previously (Chap. 2). However, the results in the two groups differed: while the post-operative mitral diastolic closure rate was less than 40 mm/sec. in only 2 patients from Group 1, it was less than 22 mm/sec. in 6 patients from Group 2.

Form of the diastolic closure movement.

Diastolic closure was biphasic in 10 pre-operative echograms.

Ultrasonic evidence of calcification.

Mitral valve calcification was diagnosed from the echograms of 9 patients in Group 1 and of 10 in Group 2.

Comparison of echocardiographic, clinical and operative findings.

Severity of stenosis

Although most of the pre-operative diastolic closure rates fell within a narrow range, they were compared with the operative assessment of the severity of mitral stenosis (Table 5,1). When the mean for each category was considered, the diastolic closure rate was found to decrease with increasing severity of stenosis. However, in individual cases the diastolic closure rate was an inaccurate index of the severity of stenosis.

Pressures were measured at operation in all Group 1 patients. Although, in general, high pressures were associated with low diastolic closure rates, the findings were variable and there were no significant correlations between the diastolic closure rate and mean pulmonary artery pressure, mean left atrial pressure or mitral end-diastolic gradient at operation.

The/...

The diastolic closure rate was also assessed against pre-operative pressures. Observations were made on 25 patients: heavily calcified valves were excluded and results from patients who were awaiting valvotomy but who were not in the present series were added. Significant negative correlations were found between the diastolic closure rate and mean pulmonary artery pressure ($r = 0.48$; $P<0.05$), pulmonary artery systolic pressure ($r = 0.45$; $P<0.025$) and mean pulmonary capillary pressure ($r = 0.51$; $P<0.02$) at rest.

Exercise pulmonary artery and pulmonary capillary pressures, which were measured in only 7 cases, did not correlate with the diastolic closure rate.

Calcification

Evidence of mitral calcification was considered to be present on 19 pre-operative echograms but in 8 cases the operative findings failed to confirm the diagnosis. At operation, 13 valves were noted to be calcified (Fig. 5,8): there was echocardiographic evidence of calcification in 11 of them. Cardiac screening was done routinely in Group 2: of the 7 valves found at operation to be calcified, all were identified by echocardiography while only 4 were detected by fluoroscopy.

Mitral regurgitation.

At operation, mild mitral regurgitation was confirmed in 9 of the 14 cases in whom it had been suspected clinically and 2 additional cases were identified. The echogram amplitudes and the diastolic closure rates in these 11 patients with confirmed mitral incompetence (Figs. 5,4 to 5,7), did not obviously differ from those of patients with competent valves. While there was evidence of calcification on the echograms of 6 patients with mitral regurgitation, the finding was clearly not restricted to such cases.

A biphasic diastolic closure movement with a short first component (Figs. 5,9 and 5,10) was observed in 6 regurgitant valves, one of which was calcified. The echograms in Fig. 5,9 were recorded from a patient who was found at closed valvotomy to have a mobile anterior cusp and a significant regurgitant jet, palpable 1 cm. behind/...

behind the mitral valve.

The patient whose echograms are shown in Fig. 5,10 was submitted to open-heart surgery in view of an additional aortic valve lesion and because cardiac catheterisation had indicated tight mitral stenosis with some regurgitation and possible calcification. At operation the aortic lesion was of insufficient severity to warrant aortic valve replacement. The mitral valve was found to be severely stenotic, slightly incompetent and moderately calcified. Decalcification, valvotomy with mobilisation of the sub-valvar apparatus and a limited annuloplasty were performed.

Cusp mobility

An operative assessment of anterior cusp "mobility" was made in 16 cases from Group 1. Mobility and echogram amplitude were broadly correlated. Thus, the amplitude of the anterior cusp echogram ranged from 23.0 to 34.0 (mean 28.7) mm in 10 mobile valves and from 20.0 to 29.0 (mean 23.2) mm in 6 valves with varying impairment of cusp mobility. In mitral regurgitation, cusp mobility determined the form of the diastolic closure movement: biphasic movements were recorded only from incompetent valves which retained some mobility.

No correlation was observed between the amplitude of the posterior cusp echogram and the surgical assessment of posterior cusp mobility. Although the amplitudes of the posterior cusp echograms in Fig. 5,11 are similar, the posterior leaflet was judged at operation to be "freely mobile" and "very immobile" in cases (a) and (b) respectively.

Subvalvar stenosis

There were no distinctive echocardiographic features in the 9 patients found to have subvalvar stenosis. In particular, their echogram amplitudes did not differ from those of patients in whom the stenosis was purely valvar.

The post-operative echogram

In most cases, the diastolic closure rate rose post-operatively (Figs. 5,6 and 5,7) but even where an excellent valvotomy was achieved in a mainly membranous valve (Fig. 5,12), it did not reach normal levels.

The/...

The echograms in Fig. 5,13 were recorded from a valve with tight stenosis and jet incompetence. Although incomplete commisurotomy and moderate subvalvar stenosis produced a residual end-diastolic gradient of 4 mm.Hg., the diastolic closure rate rose considerably with operation.

The diastolic closure rate increased least in instances where calcification and a low amplitude echogram co-existed (Fig. 5,8). Thus, while the post-operative diastolic closure rate did not exceed 30 mm/sec in the 5 calcified valves with pre-operative amplitudes below 22 mm, it ranged (with one exception) from 30 - 65 mm/sec when calcification was associated with a normal pre-operative amplitude. The echograms in Fig. 5,14 illustrate this point.

The post-operative diastolic closure rate tended to reflect the degree to which the surgeon considered stenosis had been relieved (Table 5,2).

Discussion

The results of the echocardiographic measurements in the two groups of patients have been presented separately because the groups were clinically distinct and because the surgeon who performed the Group 1 valvotomies co-operated in the study and described his operative findings in detail.

The echogram amplitudes of both normal and rheumatic mitral valves reported here are higher than those observed by others, who measured them from chart records of single echoes (Winters et al., 1967; Gustafson, 1967; Wharton and Lopez Bescos, 1970). The present findings do, however, support the general conclusion of these workers that the amplitude of the mitral anterior cusp echogram reflects the mobility of that structure.

Mobility, the resultant of varying degrees of thickening, calcification, commisural fusion and subvalvar disorganisation, is a parameter which surgeons find difficult to define but useful to assess. It can be concluded from the present results that important impairment of mobility, precluding successful valvotomy, can be anticipated when the amplitude of the anterior cusp echogram is less than 22 mm. All the abnormally low amplitude echograms recorded here were/...

were from heavily calcified valves, some with subvalvar disorganisation. However, there was no evidence relating low echogram amplitude and subvalvar stenosis in the absence of calcification.

The pre-operative diastolic closure rates in this study are at variance with the higher values, particularly for moderate stenosis, reported by Segal et al. (1966) but are in accord with the observations of Effert et al. (1964), Edler (1967) and Winters et al. (1967). These workers demonstrated a good correlation between the diastolic closure rate and the mitral valve area. Their findings are corroborated by the broad correlation observed here between the diastolic closure rate and the surgeon's operative assessment of the severity of stenosis.

It is clear that echocardiography distinguishes accurately between mild and haemodynamically significant mitral stenosis. It is rarely of practical importance to distinguish between moderate and severe stenosis; both are significant and the patient's symptoms determine the need for operative intervention. Hence, it is not a major disadvantage that in this study, as in others, these two grades of stenosis could not be consistently differentiated on the basis of the diastolic closure rate.

This investigation and that of Gustafson (1967) have demonstrated a significant inverse relation, in some stenotic valves, between the diastolic closure rate and pre-operative right heart pressures. However, presumably because cusp pliability is among the determinants of the diastolic closure rate, no such correlation exists in the presence of heavy calcification, and even in its absence, the correlation may be imprecise in individual cases. These observations have practical implications for the assessment of patients with clinically pure mitral stenosis. Thus, when ultrasound demonstrates a mobile valve, right heart catheterisation will provide no useful additional information whereas pressure measurements may be indicated to investigate the haemodynamic effects of an echocardiographically rigid valve.

It/...

It is evident that the post-operative diastolic closure rate broadly reflects the degree to which stenosis has been relieved. However, because it has a number of determinants, the diastolic closure rate remains subnormal even after an excellent valvotomy. The diastolic closure rate measured at operation (Johnson et al., 1972) can therefore only be regarded as an approximate index of the success of commisurotomy. The post-operative results in the present series are similar to those reported by Effert et al. (1964) and Edler (1967) but these workers concluded, on the basis of lower values in normal subjects, that the diastolic closure rate returned after successful valvotomy to normal or near normal levels.

Heavy mitral valve calcification, with its associated disruption of leaflet and subvalvar anatomy is likely to preclude successful valvotomy. Its pre-operative identification is important. Ideally, in such cases, the valve should be examined under direct vision on cardio-pulmonary by-pass and the decision to proceed to open valvotomy or to mitral valve replacement should then be made. Joyner et al., (1965) and Segal et al. (1966) described the echocardiographic features of the calcified mitral valve. The present findings show ultrasound to be superior to fluoroscopy in the detection of mitral calcification. In the two instances where echocardiography failed to identify calcium later demonstrated at operation, calcification was thought to be restricted to the root of the anterior commisure. Although calcification was diagnosed only if multiple echoes persisted when the intensity of the incident ultrasonic beam was minimal, there were 8 apparent false positives. Previous experience may explain the discrepancy: ultrasound identifies mild calcification which can be demonstrated microscopically but which cannot be diagnosed at open valvotomy (Benham, Rodger and Pridie, unpublished).

When an apical systolic murmur co-exists with evidence of mitral stenosis, the patient's suitability for valvotomy may be questionable. In this context, echocardiography can provide quite accurate information which renders haemodynamic investigation unnecessary in many instances. Clearly, if echocardiography demonstrates/...

demonstrates a rigid, heavily calcified valve, closed valvotomy is precluded on this basis alone and the patient is a candidate for cardio-pulmonary by-pass with a view to open valvotomy or mitral valve replacement: in these circumstances, additional cardiac catheterisation has little to commend it. If, on the other hand, the anterior cusp is pliable, ultrasound can be used to identify mitral regurgitation and to assess its significance.

Segal et al. (1966) observed biphasic diastolic closure movements in the presence of predominant mitral regurgitation. They were, however, unable to differentiate echocardiographically between pure mitral stenosis and dominant stenosis with minor regurgitation: others have had the same experience (Burgess et al., 1973). The present study shows that provided the anterior cusp is pliable, the two lesions can be distinguished. Thus, even when stenosis is dominant and severe, minor mitral incompetence is reflected in a biphasic diastolic closure movement with a short initial component. This initial component is relatively fast (14 to 66 mm/sec) but because it is of short duration, it contributes little to the mean diastolic closure rate. Hence, in assessing the significance of rheumatic mitral regurgitation, the duration of the first component of diastolic closure should be considered. Mean diastolic closure rates thus calculated from predominantly stenotic valves, fall far below the figures quoted by Segal et al. (1966).

After valvotomy, diagnostic problems could arise with echograms like the one in Fig. 5,9b, which could be interpreted as evidence of good relief of stenosis or of predominant mitral regurgitation in a pliable valve. However, the two possibilities are unlikely to be confused clinically and the echogram can still be of practical value. Thus, if such a record is obtained from a patient who is failing to improve post-operatively, it can at least be concluded that residual stenosis is not responsible.

The diastolic closure rate of a normal mitral valve may be significantly decreased in the presence of a non-compliant left ventricle. The differential diagnosis from mitral stenosis is greatly simplified by the observation that the/...

the anterior and posterior cusp echoes of a stenotic valve move in the same direction during diastole (Duchak, Chang and Feigenbaum, 1972). This finding is explicable if the relative positions of the two cusps are as shown in Fig. 5,15: it can be envisaged how the ultrasonic beam could be angled to traverse both cusps as they move in diastole in essentially the same direction towards the transducer.

In the present investigation the diagnosis of mitral stenosis was never in doubt and apart from identifying posterior leaflet calcification, the posterior cusp echogram provided no useful information. In particular, there was no evidence that posterior cusp mobility could be assessed from it.

Because, theoretically, the direction of posterior leaflet movement might be expected to return to normal after successful valvotomy, a particular effort was made to record the posterior cusp echogram post-operatively. In every case the anterior and posterior cusp echoes moved in the same direction before operation and despite successful valvotomy, the abnormality persisted in each.

Interpreted intelligently in the clinical context, echocardiography can go a long way towards replacing cardiac catheterisation in the assessment of the rheumatic mitral valve. In essence it provides direct information on the structure and function of the valve, from which the haemodynamic effects can be implied. Its scope is increasing as ultrasound measurements of left ventricular size and performance are validated. It therefore makes economic sense that cardiologists should become familiar with details of the method, its applications and limitations.

Summary

Mitral echograms were recorded before (37 patients) and again after (35 patients) mitral valvotomy. Following operation, echogram amplitude was unchanged and the mitral diastolic closure rate increased but remained subnormal.

The following conclusions were reached from a comparison of the operative and the echocardiographic findings:- 1) While mild and haemodynamically significant mitral/...

mitral stenosis can be distinguished, moderate and severe stenosis cannot be differentiated on the basis of the diastolic closure rate. 2) Provided that the cusps are pliable, pure mitral stenosis can be differentiated echocardiographically from predominant stenosis with minor regurgitation. 3) Anterior but not posterior cusp echogram amplitude is an index of cusp mobility. 4) There are no echocardiographic features diagnostic of subvalvar stenosis. 5) Ultrasound is superior to fluoroscopy in the detection of mitral calcification. 6) Ultrasound differentiates reliably between patients suitable for closed mitral valvotomy and those requiring open valvotomy or mitral valve replacement.

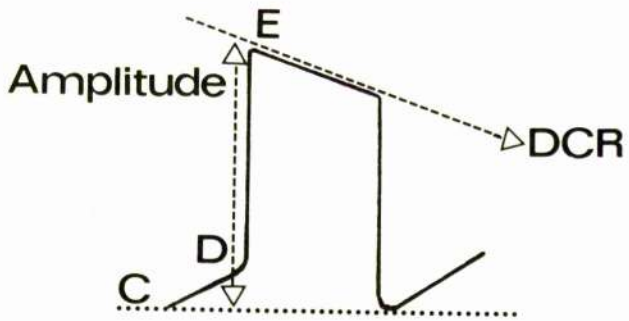


FIG. 5,1 METHODS OF MEASUREMENT

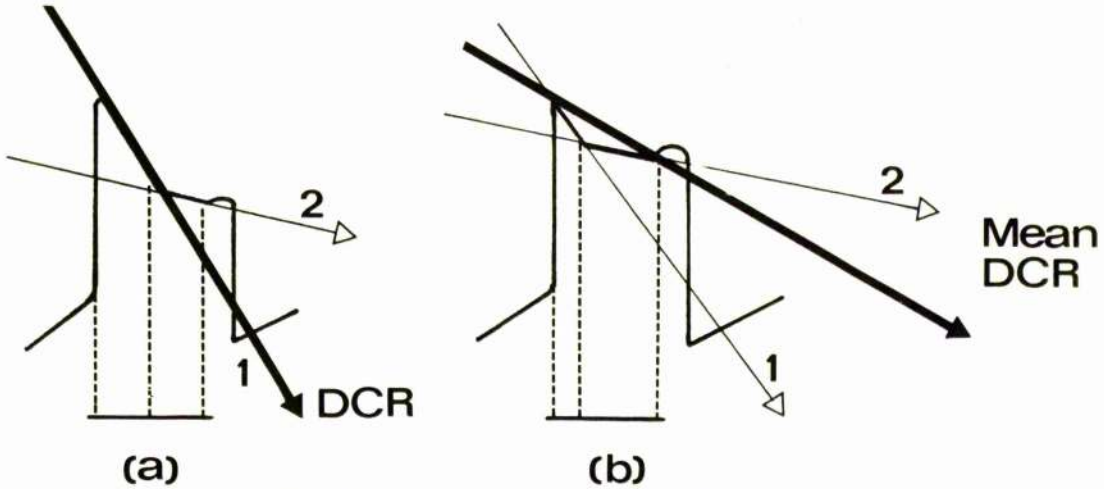


FIG. 5,2 METHODS OF MEASUREMENT - See Text

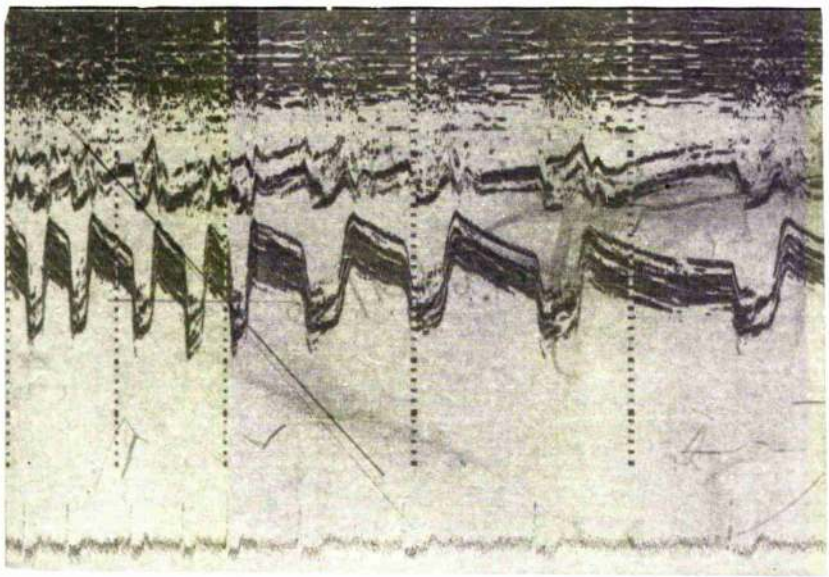


FIG. 5,3 MITRAL CALCIFICATION

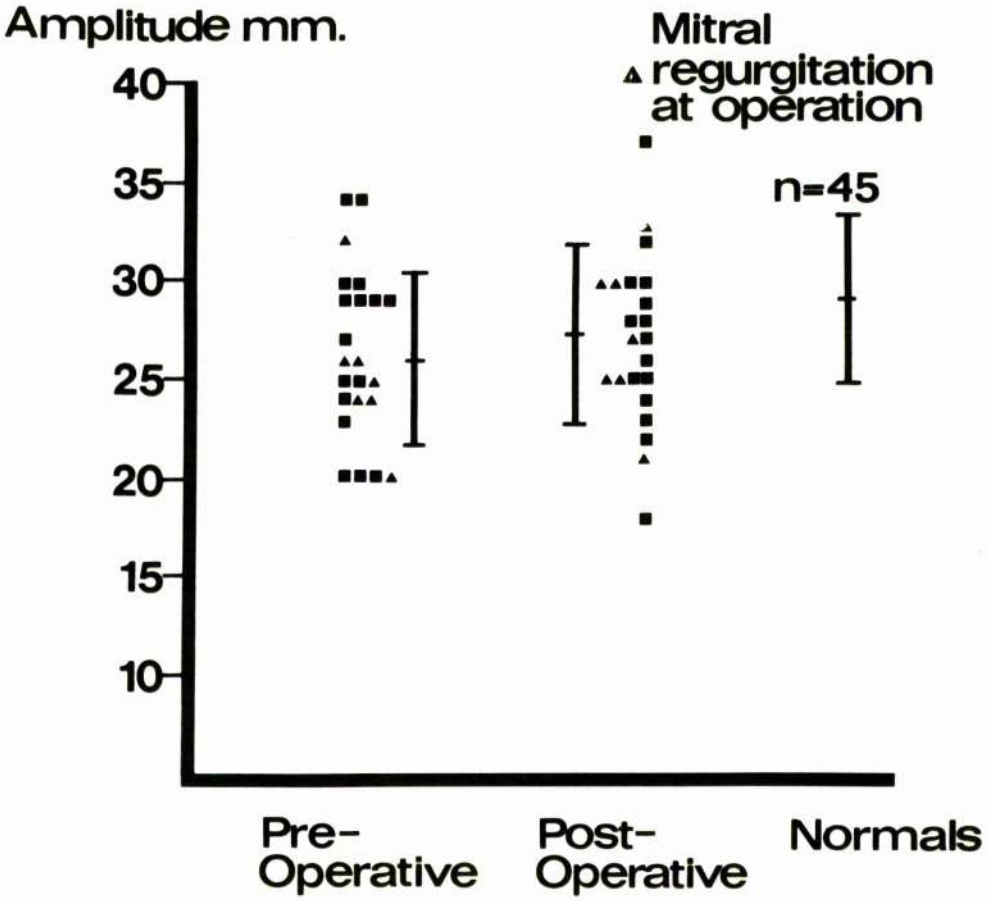


FIG. 5,4 AMPLITUDE - GROUP 1

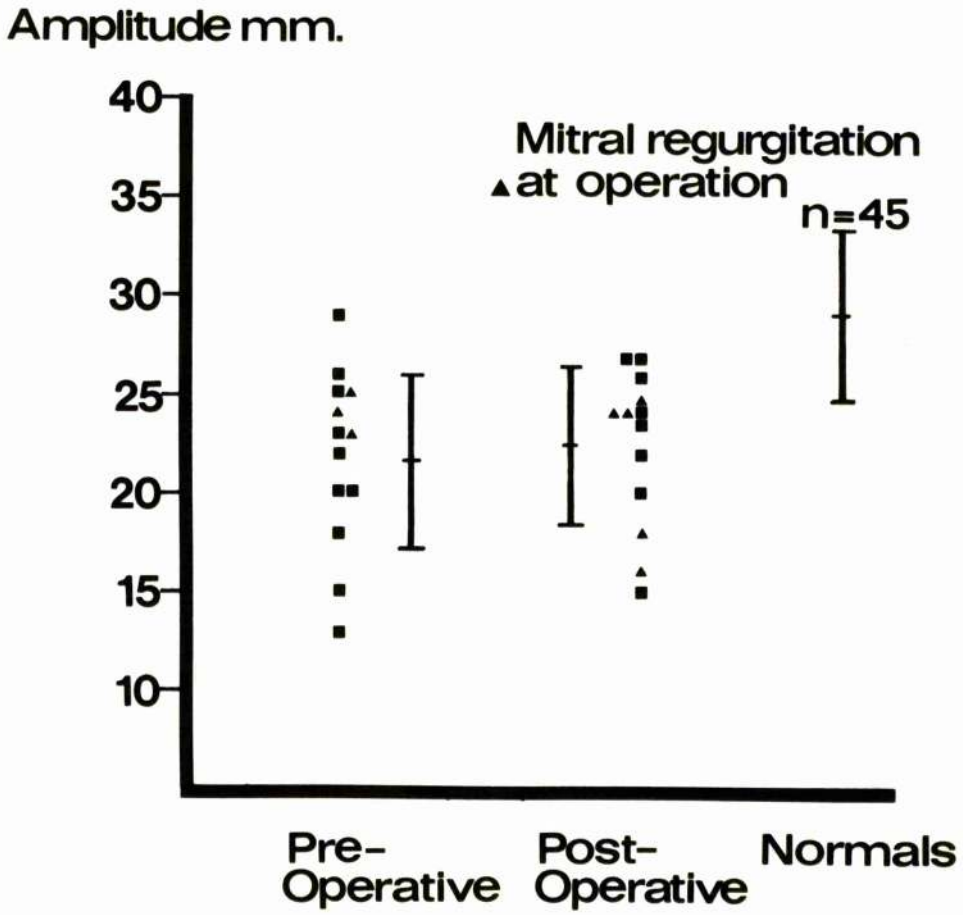


FIG. 5,5 AMPLITUDE - GROUP 2

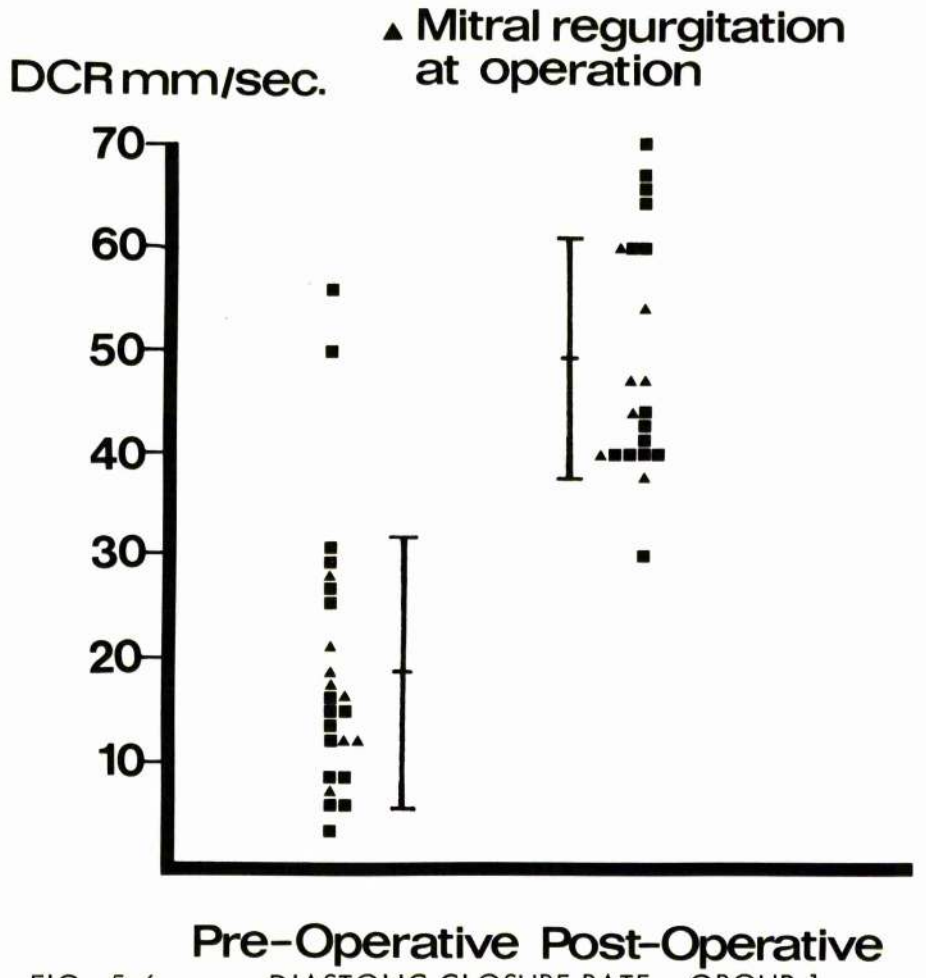


FIG. 5,6 DIASTOLIC CLOSURE RATE - GROUP 1

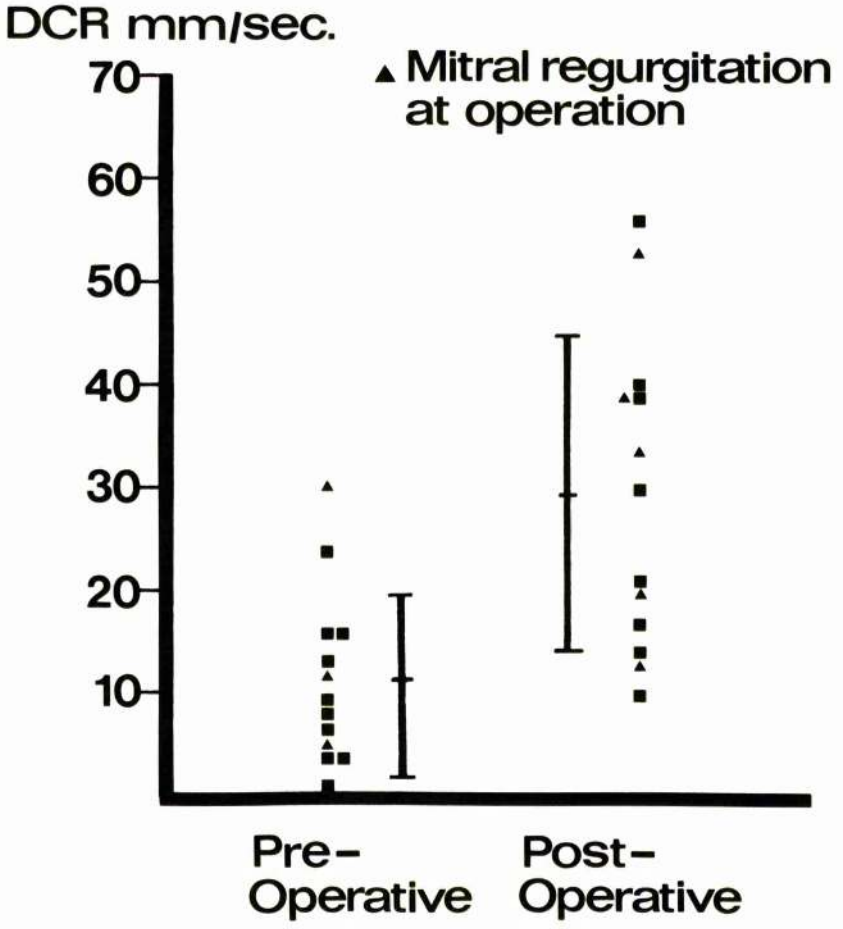


FIG. 5,7 DIASTOLIC CLOSURE RATE - GROUP 2

TABLE 5,1 SEVERITY OF STENOSIS AT OPERATION AND THE PRE-OPERATIVE DCR

Severity of Stenosis	Number of Patients	Pre-operative DCR mm/sec. Range	Mean
Group 1			
Mild	3	18 - 56	35
Moderate	12	6 - 50	20
Severe	9	3 - 28	13
Group 2			
Moderate	5	1 - 30	17
Severe	8	4 - 16	8

TABLE 5,2

RELIEF OF STENOSIS AND THE POST-OPERATIVE DCR

Quality of Valvotomy	Number of Patients	Post-operative DCR mm/sec. Range	Mean
Group 1			
Excellent	3	43 - 70	58
Good	10	40 - 66	50
Fair	5	38 - 67	48
Poor	3	30 - 54	41
Group 2			
Good	5	17 - 56	37
Fair	6	10 - 53	28
Poor	2	13 - 20	17

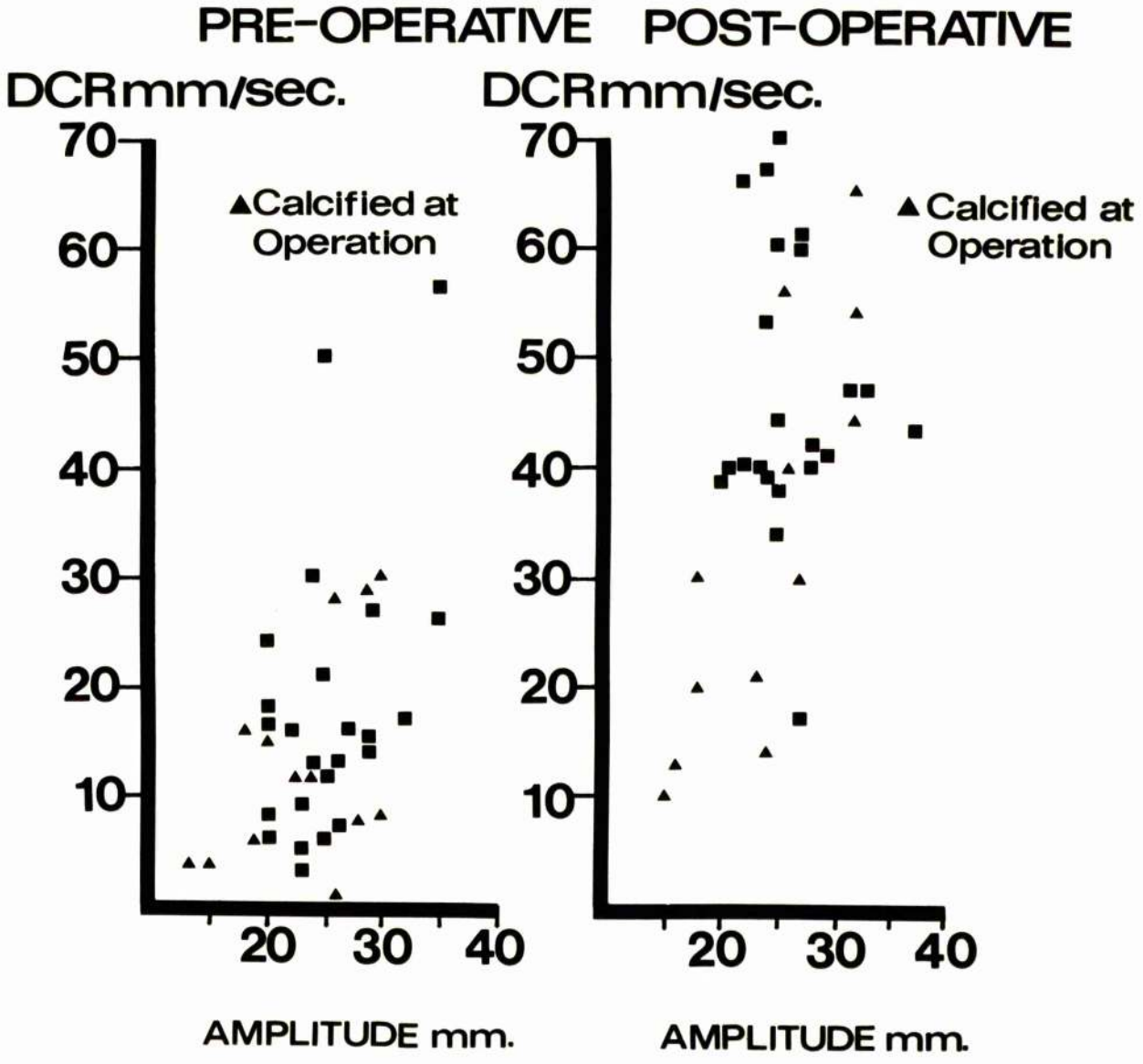
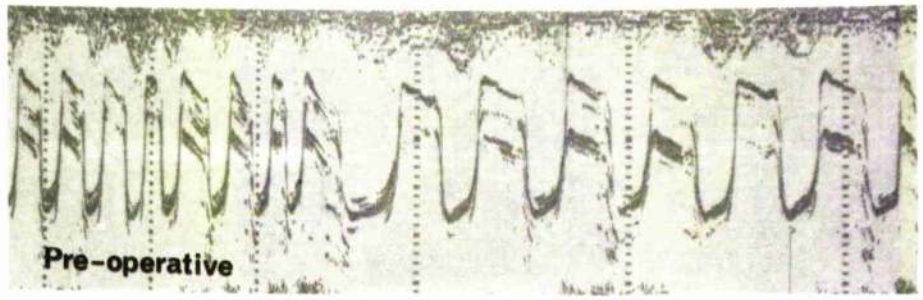
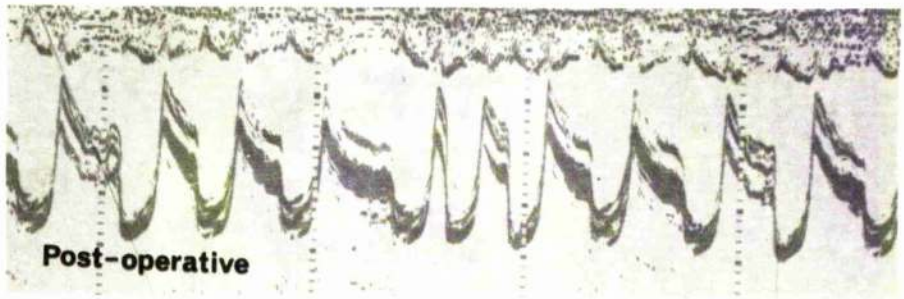


FIG. 5,8 COMPARISON OF ECHOCARDIOGRAPHIC MEASUREMENTS IN CALCIFIED AND NON-CALCIFIED VALVES.

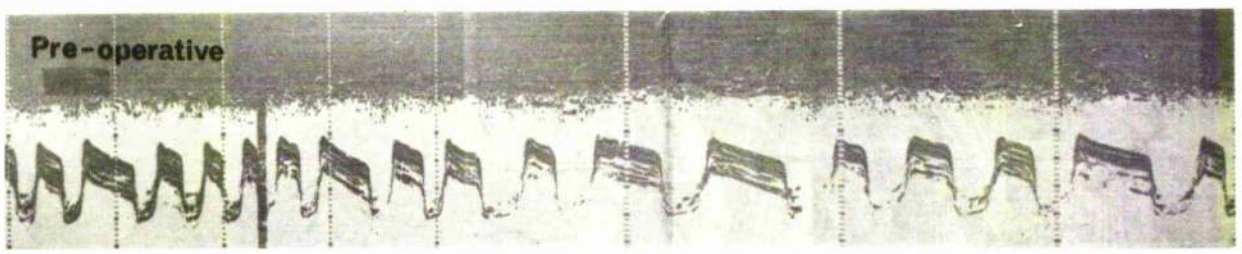


(a)

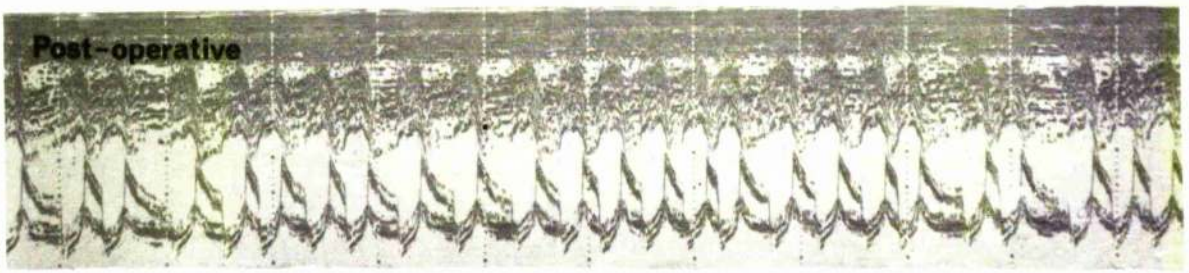


(b)

FIG. 5,9 MILD MITRAL REGURGITATION IN A MOBILE, PLIABLE VALVE PRE-OPERATIVE AMPLITUDE = 32 mm AND DCR = 17 mm/sec.



(a)



(b)

FIG. 5,10 MILD MITRAL REGURGITATION IN A MOBILE, CALCIFIED VALVE PRE-OPERATIVE AMPLITUDE = 30 mm AND DCR = 13 mm/sec.

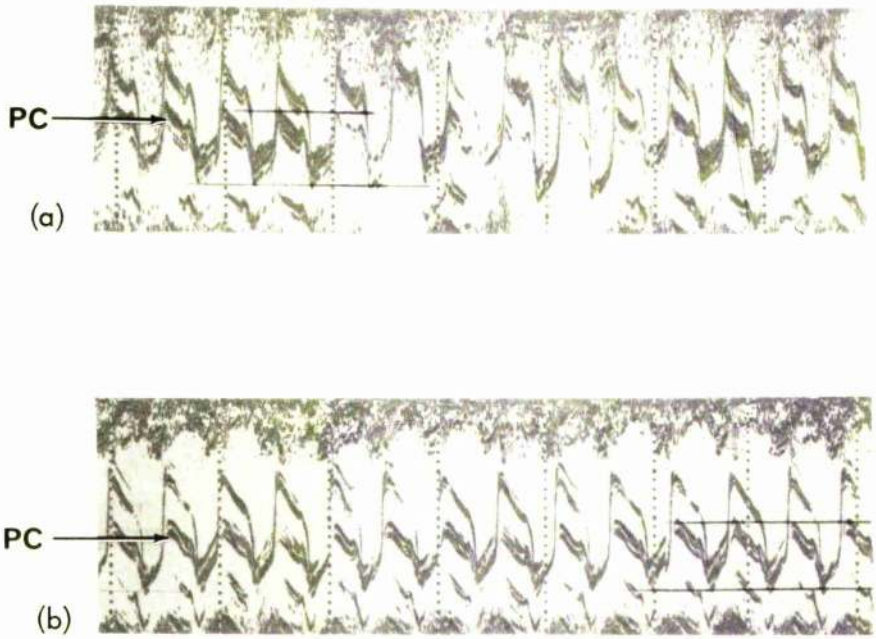


FIG. 5,11 POSTERIOR CUSP ECHOGRAM AMPLITUDE = 17.5 mm IN (a) AND 16.5 mm IN RECORD (b)

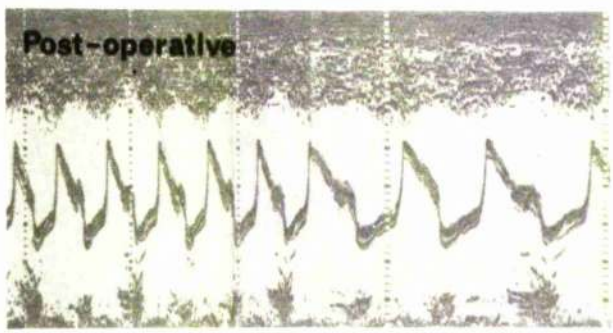


FIG. 5,12 DCR = 43 mm/sec.

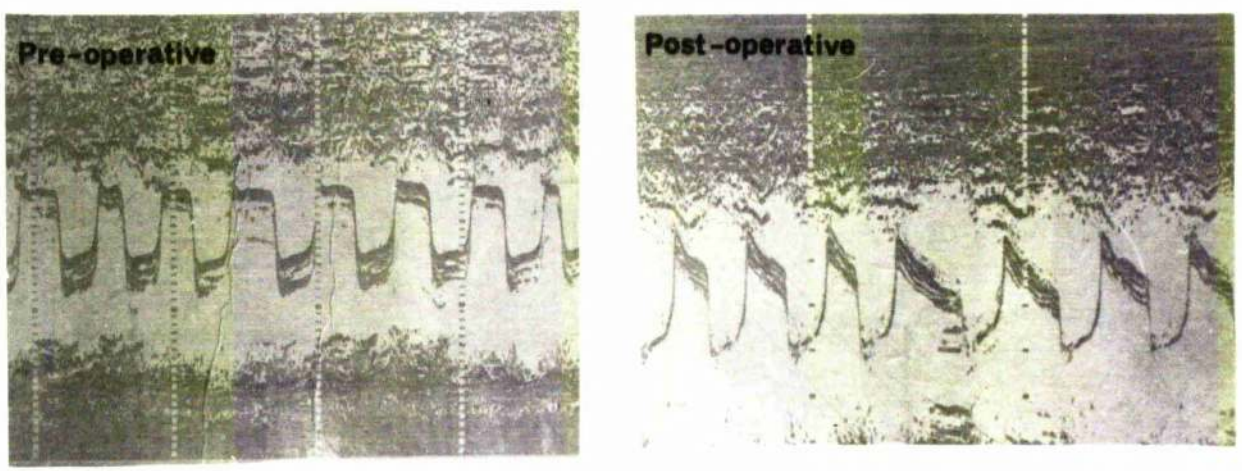
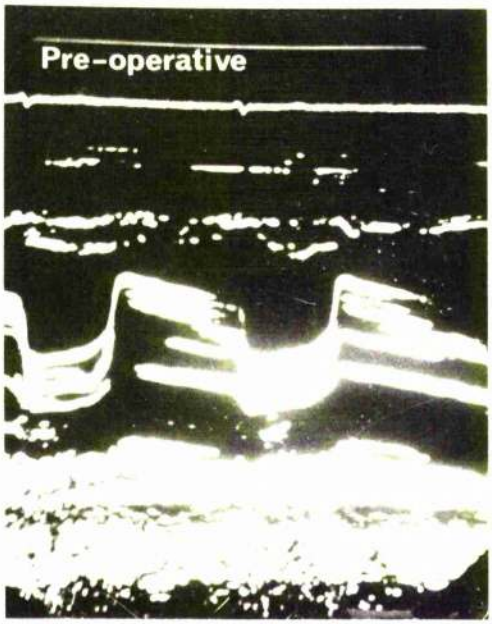
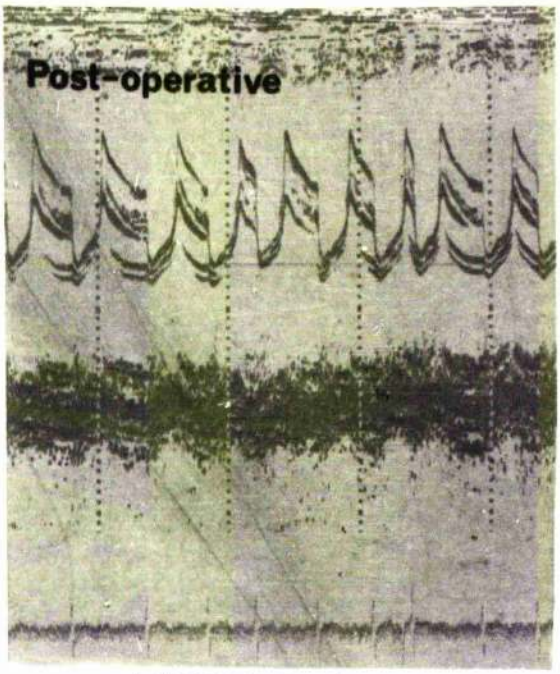


FIG. 5,13 DCR = 7 mm/sec. BEFORE AND 60 mm/sec. AFTER OPERATION

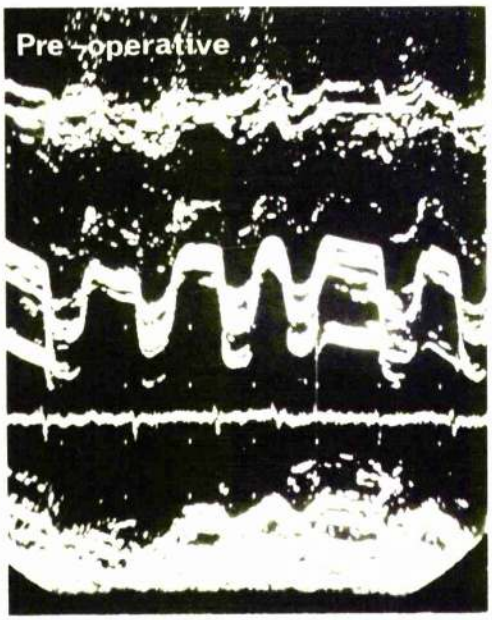


(a) AMPLITUDE = 26 mm
DCR = 11 mm/sec.



AMPLITUDE = 26 mm
DCR = 56 mm/sec.

(b) AMPLITUDE = 20 mm
DCR = 5 mm/sec.



AMPLITUDE = 21 mm
DCR = 17 mm/sec.

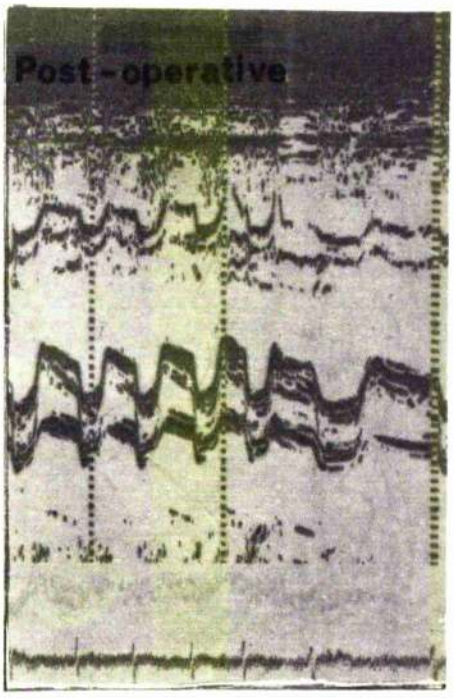
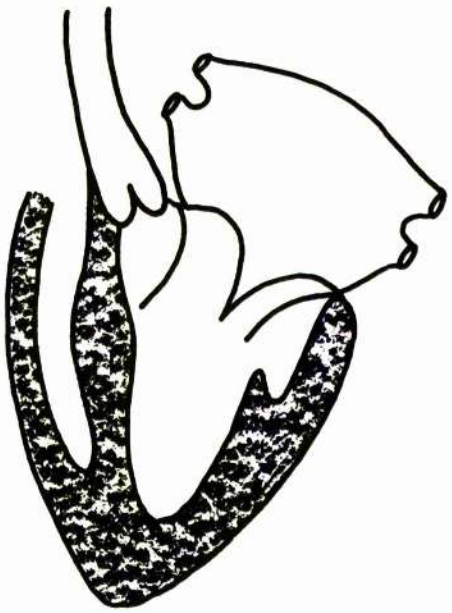
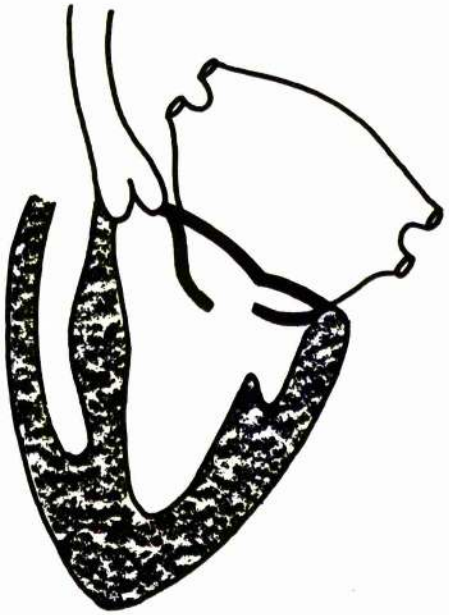


FIG. 5,14 RECORDS BEFORE AND AFTER CLOSED VALVOTOMY IN TWO PATIENTS WITH MITRAL CALCIFICATION. ECHOGRAM AMPLITUDE IS NORMAL IN CASE (a) AND OPERATION WAS SUCCESSFUL.



NORMAL



RHEUMATIC MITRAL VALVE

FIG. 5,15 THE DIRECTION OF CUSP MOVEMENT IN THE RHEUMATIC MITRAL VALVE.

CHAPTER 6

THE MITRAL ECHOGRAM IN AORTIC VALVE DISEASE

The features of the mitral echogram in aortic valve disease have been described (Winsberg and Mercer, 1971; Pridie, Benham and Oakley, 1971). In patients with aortic regurgitation, functional and organic mitral diastolic murmurs can be differentiated by ultrasound (Pridie et al., 1971). The mitral diastolic closure rate in aortic regurgitation has been found to be related to the left ventricular stroke volume (Ziady et al., 1973). There is evidence that reduced left ventricular compliance is reflected in a slowing of the diastolic closure rate of the non-rheumatic valve (Popp and Harrison, 1969; Layton et al., 1973). In theory, therefore, the mitral diastolic closure rate should provide an index of the severity of isolated aortic stenosis. These observations suggest that, ultrasonic assessments of left ventricular size and performance apart, echocardiography of the mitral valve can by itself make an important contribution to the investigation of patients with predominant aortic valve disease.

The first aim of the present study was to investigate the reliability of mitral valve echocardiography both in the indirect assessment of the aortic lesion and in the direct assessment of the mitral valve in patients with predominant aortic valve disease. The second aim was to compare the contributions of clinical, haemodynamic and ultrasonic investigations and thus to review the role of cardiac catheterisation in the assessment of patients with predominant aortic valve lesions.

Patients (Tables 6,1, 6,2 and 6,4)

54 patients with predominant aortic valve disease were studied. 32 of them underwent haemodynamic investigation including cineangiography (Tables 6,1 and 6,2). Combined left and right heart catheterisation was carried out in 26 cases and left heart catheterisation in 6. In 20 instances the aortic valve could not be traversed (Table 6,1): direct cardiac puncture was performed in 5 cases at the same procedure and in 3 at a separate procedure.

19/...

19 patients, all of whom had undergone full haemodynamic investigation, were referred for operation: 18 had open heart surgery and one had a closed mitral valvotomy (Tables 6,1 and 6,2).

Methods

The mitral anterior cusp echogram was recorded using the techniques and equipment previously described (Chap. 2). The diagnostic criteria for mitral calcification and regurgitation and the methods of measurement of the amplitude and the diastolic closure rate were as before (Chaps. 2 and 5). Fig. 6,1 shows how systolic closure of the mitral anterior cusp was timed with reference to the onset of the QRS complex of the electrocardiogram.

In the patients (1 - 32) who were catheterised, ultrasonic, clinical, haemodynamic and, where applicable, operative assessments were compared. In those who were not catheterised (33 - 54), the ultrasonic and clinical assessments were compared. For this purpose, stenosis, regurgitation and calcification of the aortic and mitral valves were graded as follows:- grade 0 - absent; grade 1 - trivial; grade 2 - mild; grade 3 - moderate; grade 4 - severe.

Results

I. Assessment of the mitral valve

1) Patients 1 - 32

Clinical assessment (Table 6,2)

In 3 patients there was conflicting evidence and clinical assessment of the mitral valve was impossible. The mitral valve was thought to be normal in 11 cases including two (1 and 21) presumed to have Austin Flint murmurs. A diagnosis of isolated mitral regurgitation was made in one patient and in 18 cases the mitral valve was judged to be rheumatic, the lesion being purely stenotic in 8 of them.

Catheter assessment (Table 6,2)

A full evaluation of the mitral valve was not always possible. Thus, failure to enter the left ventricle precluded measurement of the end-diastolic gradient/...

gradient (patients 22 - 32) and precluded angiographic assessment of mitral regurgitation in 5 instances where adequate left ventricular opacification was not achieved from an aortic root injection. Further, the right heart was not catheterised and no attempt was made to investigate the mitral valve in 6 patients in whom its normality was not clinically in doubt.

Because the end-diastolic gradient could not be measured and because the indirect left atrial pressure was technically difficult to record or had an equivocal waveform, it was impossible to confirm or exclude mitral stenosis in 6 cases, including two (patients 22 and 26) in whom no clinical diagnosis had been reached. Despite catheterisation of the left heart, an equivocal result was also obtained in patient 2 who was thought clinically to have mitral stenosis.

Mitral calcification was identified in two cases.

Ultrasonic assessment (Table 6,2)

Mitral stenosis could be confidently confirmed or excluded in 30 patients. In one case (22), the diagnosis of trivial stenosis was tentative while in another (12), the echogram was technically unsatisfactory and thus inconclusive.

Mitral regurgitation was diagnosed from the echograms of 4 patients and its presence could not be excluded in 3 others with ultrasonic evidence of moderate or heavy mitral calcification (patients 10, 11 and 20).

Mitral calcification was diagnosed in 9 cases.

Operative assessment (Table 6,2)

The operative findings are described in Table 6,2.

Comparison of catheter and ultrasonic assessments (Table 6,3)

In 19 patients, the accuracy of the catheter and ultrasonic assessments were compared in the light of the operative findings (Table 6,3).

Echocardiography was clearly superior to catheterisation in the assessment of mitral stenosis. Neither method was ideal for the investigation of mitral incompetence; it was possible to miss important regurgitation using ultrasound (patient 5), mild regurgitation using angiography (patient 24) and trivial regurgitation/...

regurgitation using both methods (patient 4). Further, in two instances an angiographic diagnosis of significant mitral regurgitation was not confirmed at operation (patients 3 and 22). Fluoroscopy failed to detect mitral calcification in two cases while in two others, the echocardiographic diagnosis of calcification was not confirmed.

In the 13 patients who were not operated, the findings at catheterisation and ultrasonic examination were broadly in accord. It is, however, interesting to note that moderate mitral stenosis with trivial regurgitation was diagnosed at catheter in a patient (9) with an unequivocally normal echogram and that there was echocardiographic evidence of heavy mitral calcification in two patients (10 and 11) with negative fluoroscopy.

2) Patients 33 - 54 (Table 6,4)

The clinical and ultrasonic assessments of the mitral valve in the patients who were not catheterised are detailed in Table 6,4. The results are widely discrepant; ultrasound confirmed the clinical diagnosis of a normal or of a rheumatic mitral valve in only 8 instances.

Accuracy of clinical assessment (Patients 1-54)

Combining the results from all 54 patients and assuming the ultrasonic diagnosis to be correct in the cases where it was not feasible to confirm it, the clinical assessment of the mitral valve was seriously in error in 15 instances (28%).

II Assessment of the aortic valve

Comparison of clinical and catheter assessments (Patients 1 - 32)

The clinical and catheter assessments of the aortic valve are detailed in Table 6,1.

Full haemodynamic investigation of the aortic valve was possible in patients 1 to 20. Although the clinical and catheter estimates of the severity of aortic stenosis were in precise accord in only 6 cases, the findings were never seriously discrepant. Thus, trivial or mild stenosis was suspected clinically in 6 patients without an aortic systolic gradient, while clinical examination/...

examination over-estimated the degree of stenosis by one grade in 5 patients and under-estimated it by the same margin in 3 others.

Estimates of the severity of aortic regurgitation clinically and at cardiac catheterisation were in accord in 20 instances. While aortic regurgitation was over-estimated clirically in 8 cases and under-estimated in 4, assessments did not differ by more than one grade.

The mitral diastolic closure rate

Technically satisfactory echograms from which the mitral diastolic closure rate could be measured accurately (Chap. 2) were recorded in 21 patients with an isolated aortic lesion.

The relation between the mitral diastolic closure rate and the severity of isolated aortic valve disease, as judged clinically or at haemodynamic investigation, was studied in 21 patients (Table 6,4). In general, the diastolic closure rate increased and decreased with increasing dominance and severity of aortic regurgitation and of aortic stenosis respectively. However, diastolic closure rates within the normal range were on occasion associated with severe regurgitation (patient 44) and with severe stenosis (patient 17).

In 7 patients with major aortic regurgitation, the mitral diastolic closure rate and the left ventricular end-diastolic pressure were found to be unrelated. Thus, the diastolic closure rate was 338 mm/sec in a patient (1) with an end-diastolic pressure of 7 mm Hg. and 255 mm/sec in a patient (7) in whom left ventricular end-diastolic pressure was 30 mm Hg. Similarly, no relation was observed between the mitral diastolic closure rate and the height of the left ventricular end-diastolic pressure in 3 patients with moderate or severe aortic stenosis.

Systolic closure of the mitral valve

Systolic closure of the mitral anterior cusp was timed in 33 patients. The results were compared with those from normal subjects, patients with isolated/...

isolated mitral stenosis, and patients with acute myocardial infarction complicated by left ventricular failure (Fig. 6,2).

In the normal subjects, closure was complete 0.06 to 0.10 sec after the onset of electrical systole. On this basis, closure was premature in 16 patients with isolated aortic valve disease. Premature closure was a constant finding when chronic aortic regurgitation was severe and a usual finding when it was moderate. Closure preceded the Q wave of the electrocardiogram only when prolongation of the PR interval or atrial fibrillation co-existed. Closure was grossly premature (0.20 sec before the Q wave) in the only patiert in the series with acute aortic regurgitation. Although closure was usually normal in the presence of predominant aortic stenosis, it occurred prematurely in two cases with PR prolongation and in one of these was complete before the onset of the QRS complex.

Closure was delayed in isolated mitral stenosis but occurred normally after successful valvotomy and in patients with combined aortic and mitral valve disease.

In acute myocardial infarction with radiographic evidence of left heart failure, there was no tendency to premature closure. It should, however, be noted that the heart rate exceeded 80 beats/min and atrio-ventricular conduction was normal in all these patients.

Discussion

It is generally accepted that clinical assessment of the mitral valve is difficult in the presence of aortic valve disease. It is therefore not surprising that mitral diastolic murmurs were seriously misconstrued in 28% of the present series or that no clinical diagnosis was recorded in a further 11%. The shortcomings of cardiac catheterisation in this context are, however, perhaps less widely appreciated. This study has shown that in patients with aortic valve disease, haemodynamic assessment of the mitral valve is both technically difficult and unreliable. Further, it has demonstrated the clear superiority of echocardiography in the investigation of the mitral valve in

such/...

such patients.

It can be concluded that echocardiography of the mitral valve should be a routine part of the investigation of patients with aortic valve disease. Provided that the recorded echogram is technically satisfactory, the method unequivocally distinguishes a rheumatic from a non-rheumatic mitral valve. With care, errors of interpretation can be eliminated but Fig. 6,3 illustrates how they may arise. Thus, although fine diastolic oscillations are present, mitral stenosis might be mistakenly diagnosed from the echogram in Fig. 6,3a. However, in Fig. 6,3b from the same patient, the mitral posterior cusp has been recorded and can be seen to oscillate and to move away from the anterior cusp during diastole: mitral stenosis is thus excluded (Duchak, Chang and Feigenbaum, 1972). The tracing in Fig. 6,3a is misleading because it is technically unsatisfactory in that the full amplitude of the anterior cusp echogram has not been recorded.

This study has shown echocardiography of the mitral valve to be, in general, unhelpful in the assessment of an aortic valve lesion.

In all their patients with aortic regurgitation, Ziady et al. (1973) observed an abnormally rapid mitral diastolic closure rate which they were able to relate to left ventricular stroke volume. However, in the present series, while the most severe grades of regurgitation were associated with the higher diastolic closure rates, these rates were not always abnormal (Table 6,5). Further, the mitral diastolic closure rate and left ventricular end-diastolic pressure were found to be unrelated.

Similarly, although the lowest diastolic closure rates were recorded from patients with significant aortic stenosis (Table 6,5), the diastolic closure rate was within the normal range in patients 16 and 17 who had aortic systolic gradients of 75 and 120 mm Hg. respectively.

It should be noted firstly that it may be difficult to record a technically satisfactory mitral echogram in the presence of aortic regurgitation and secondly that accurate measurement of the diastolic closure rate of an oscillating/...

oscillating mitral anterior cusp may be impossible. For these reasons mitral diastolic closure rates were obtained in only 21 of the non-rheumatic valves in this series.

The present evidence indicates that no valid conclusions about the severity of either aortic regurgitation or aortic stenosis can be drawn from the diastolic closure rate of the mitral valve.

The observations of Pridie et al. (1971) on mitral systolic closure in acute aortic regurgitation and in chronic regurgitation with prolongation of the PR interval have been confirmed here. Their criteria for the recognition of premature closure were extended and on this basis, premature mitral systolic closure was found to be a constant feature of severe chronic aortic regurgitation. However, the measurement of mitral systolic closure times was found to be of only limited practical value; accurate differentiation of moderate from mild aortic regurgitation was not possible.

Although echocardiography of the mitral valve contributes little to the assessment of the aortic valve, other ultrasonic techniques may have more to offer. Thus, ultrasonic estimates of the size, performance and filling characteristics of the left ventricle (Chap. 8) could obviously be useful in this context. Further, it may be possible with modern recording methods to increase the accuracy of the information obtained from the aortic valve echogram (Winsberg and Mercer, 1972).

Clinical assessment of the aortic valve proved to be acceptably accurate in this series. When the clinical examination, the electrocardiogram and the plain chest radiograph were taken into account, the severity of stenotic and of regurgitant lesions was never seriously misjudged (Table 6.1).

In line with the observations of Batson, Urquhart and Sideris (1972), aortic valve calcification identified at fluoroscopy was found to be good evidence of important aortic stenosis. It was moreover, good evidence that direct cardiac puncture would be required for full haemodynamic investigation of the aortic valve; in the present study only one such calcified valve was traversed/...

traversed.

In this series, 32 patients underwent haemodynamic investigation. It entailed 41 hospital admissions for 42 separate procedures (excluding coronary arteriography) which often yielded imprecise or incomplete information and added little to the clinical and echocardiographic findings. Cardiac catheterisation is potentially hazardous and is expensive both in terms of time and of finance. It should thus be undertaken only when it will clearly provide additional data relevant to the patient's management: its role in the investigation of patients with aortic valve disease merits re-appraisal.

It is well established that patients with major aortic valve lesions may be asymptomatic until haemodynamic compensation fails. In the absence of decompensation and of symptoms, they are therefore referred for cardiac surgery on the basis of an assessment of the severity of their lesion. In adults this assessment can usually be made with acceptable accuracy at the bedside: when the clinical evidence is good, cardiac catheterisation simply to corroborate it, is not justifiable. When there is nothing to suggest that the lesion is other than valvar and particularly when there is positive evidence that it is valvar e.g. aortic valve calcification or a co-existing rheumatic mitral valve, there is no need to demonstrate the site of the lesion. Haemodynamic investigation of the mitral valve is unnecessary because echocardiography provides the surgeon with adequate information on its structure and function.

Although it has been argued here that routine cardiac catheterisation is frequently redundant in the investigation of patients with aortic valve disease, the same does not apply to coronary arteriography and left ventricular function studies. Thus, if there is a history of angina, if the ultrasonic left ventricular ejection fraction is reduced or if impaired myocardial performance is suspected on other grounds, coronary arteriography and left ventricular angiography are clearly indicated.

A more rational deployment of investigative resources could simplify and at the same time improve the pre-operative assessment of patients with aortic valve disease.

Summary

Mitral echograms were recorded in 54 patients with predominant aortic valve disease. Of these, 32 were catheterised and 19 were operated. No patient was referred for cardiac surgery without prior haemodynamic investigation.

The catheter and ultrasound assessments of the mitral valve were compared and their accuracy was investigated in the light of the operative findings. While catheterisation provided an adequate assessment of the mitral valve in only 17 of the 32 patients, rheumatic mitral valve disease was confidently confirmed or excluded by ultrasound in 30 of them. From the operative findings it was concluded that in the context of dominant aortic valve disease, echocardiography is superior to cardiac catheterisation for the diagnosis of mitral stenosis and that mitral regurgitation may be overlooked by either method.

In 21 patients with isolated aortic lesions, the mitral diastolic closure rate tended to increase and decrease with increasing dominance and severity of aortic regurgitation and aortic stenosis respectively but it was of no diagnostic value in individual cases. Further, no relation was observed between the mitral diastolic closure rate and the left ventricular end-diastolic pressure in severe aortic regurgitation. Although premature systolic closure was invariable in severe and usual in moderate aortic regurgitation, mild and moderate regurgitation could not be differentiated on this basis. It was concluded that the mitral echogram is rarely helpful in the assessment of an aortic valve lesion.

Clinical assessment of the mitral valve is unreliable in the presence of aortic valve disease. No clinical diagnosis was reached in 11% of the present series and the clinical assessment, as judged by the echocardiographic findings, was seriously in error in a further 28%.

TABLE 6,1 ASSESSMENT OF THE AORTIC VALVE

		Clinical diagnosis		Catheter diagnosis			Operative diagnosis			Aortic valve	Aortic valve	Cardiac
		AS	AR	AS	AR	Ca	AS	AR	Ca	Surgery	traversed	puncture
1	A.B.	0	4	0	4	0	0	4	0	AVR	+	−
2	E.D.	0	4	0	4	3	0	4	3	AVR	+	−
3	A.G.	0	3	1	3	0	0	3	0	AVR	+	−
4	C.M.	1	2	0	2	0	1	2	0		+	−
5	P.C.	1	2	0	2	0	3	3	0	AVR	+	−
6	M.C.	2	3	0	3	0	0	2	0		+	−
7	S.L.	1	4	0	4	0					+	−
8	T.S.	0	3	0	3	0					+	−
9	W.McK.	2	3	2	2	0					+	−
10	W.W.	1	1	2	1	0					+	−
11	M.R.	1	3	0	2	0					+	−
12	J.R.	1	4	0	3	0					+	−
13	J.S.	2	2	2	2	1	3	?	0	AVR	−	+
14	J.W.	3	4	4	3	3	3	3	3	AVR	−	+
15	A.T.	4	0	3	1	4	3	0	4	AVR	−	+
16	A.B.	4	1	3	0	0	3	0	0	Valvotomy	−	+
17	J.R.	4	1	4	1	4	4	0	4	AVR	−	+
18	M.T.	4	2	3	3	2	4	2	4	AVR	−	+
19	E.B.	3	2	2	3	0					−	+
20	R.H.	3	2	2	1	3					−	+
21	A.A.	0	4		4	4	2	4	3	AVR	−	−
22	E.F.	4	3		4	4	3	3	4	AVR	−	−
23	H.B.	4	2		2	4	4	2	4	AVR	−	−
24	A.M.	2	2		2	0		1	0		−	−
25	D.C.	3	3		3	0	3	3	2	AVR	−	−
26	J.C.	2	4		3	4	2	3	4	AVR	−	−
27	A.P.	3	3		3	4	4	3	4	AVR	−	−
28	M.W.	2	2		2	0					−	−
29	E.T.	2	2		2	0					−	−
30	M.B.	2	2		2	1					−	−
31	B.McK.	2	3		3	4					−	−
32	R.G.	2	3		2	3					−	−

TABLE 6,2 ASSESSMENT OF THE MITRAL VALVE

	Clinical diagnosis		Catheter diagnosis			Ultrasound diagnosis			Operative diagnosis			Mitral valve surgery
	MS	MR	MS	MR	Ca	MS	MR	Ca	MS	MR	Ca	
1	0	0	*	0	0	0	0	0	0	0	0	
2	3	0	?	0	0	0	0	0	0	0	0	
3	0	3	0	3	0	0	0	0	0	0	0	
4	4	0	3	0	0	0	0	2	4	1	0	closed valvotomy
5	4	2	3	3	0	4	0	2	3	3	2	open valvotomy
6	4	2	4	2	1	4	2	2	4	2	3	open valvotomy
7	0	0	0	2	0	4	0	0				
8	0	0	*	0	0	0	0	0				
9	2	2	3	1	0	0	0	0				
10	4	0	4	1	0	0	?	4				
11	3	0	4	0	0	4	?	4				
12	2	4	0	0	0	4	?	0				
13	3	0	2	0	0	2	0	0	3	0	0	open valvotomy
14	2	2	0	0	0	0	0	0	0	0	0	
15	0	0	0		0	0	0	0	0	0	0	
16	0	0	0	0	0	0	0	0	0	0	0	
17	0	0	*		0	0	0	0	0	0	0	
18	0	0	0	0	0	0	0	0	0	0	0	
19	?	0	2	0	0	3	2	0				
20	3	2	2		3	4	?	3				
21	0	0	*	0	0	0	0	0	0	0	0	
22	?	0	?	2	0	1	0	0	0	0	0	
23	3	0	?	1	0	4	0	0	3	1	0	MVR
24	3	2	?	0	0	3	2	0	3	2	0	open valvotomy
25	3	2	?	0	0	4	0	2	3	0	0	open valvotomy
26	?	0	?	0	0	4	0	1	4	0	3	MVR
27	0	0	*	0	0	0	0	0	0	0	0	
28	3	3	3		0	1	3	2				
29	3	0	2		0	3	0	0				
30	3	0	3	0	0	4	0	0				
31	0	0	?	0	0	0	0	0				
32	0	0	*	0	0	0	0	0				

* No right heart catheter

TABLE 6,3 ACCURACY OF CLINICAL AND ULTRASONIC ASSESSMENTS IN 19 OPERATED PATIENTS

	Mitral Stenosis		Mitral Regurgitation		Calcification	
	Catheter	UCG	Catheter	UCG	Catheter	UCG
1		✓	✓	✓	✓	✓
2	?	✓	✓	✓	✓	✓
3	✓	✓	X	✓	✓	✓
4	✓	✓	X	X	✓	X
5	✓	✓	✓	X	X	✓
6	✓	✓	✓	✓	✓	✓
13	✓	✓	✓	✓	✓	✓
14	✓	✓	✓	✓	✓	✓
15	✓	✓		✓	✓	✓
16	✓	✓	✓	✓	✓	✓
17		✓		✓	✓	✓
18	✓	✓	✓	✓	✓	✓
21		✓	✓	✓	✓	✓
22	?	X	X	✓	✓	✓
23	?	✓	✓	X	✓	✓
24	?	✓	X	✓	✓	✓
25	?	✓	✓	✓	✓	X
26	?	✓	✓	✓	X	✓
27		✓	✓	✓	✓	✓

TABLE 6,4 COMPARISON OF CLINICAL AND ULTRASONIC ASSESSMENTS IN PATIENTS NOT CATHETERISED

		Clinical Diagnosis				Ultrasound Diagnosis		
		AS	AR	MS	MR	MS	MR	Ca
33	R.M.	2	3	?	0	0	0	0
34	S.M.	3	3	?	0	0	0	0
35	A.B.	3	2	?	0	0	0	0
36	J.H.	0	4	0	0	0	0	0
37	J.E.	1	4	2	0	0	0	0
38	J.G.	3	2	2	0	0	0	0
39	M.S.	2	2	3	0	0	0	0
40	L.B.	4	2	2	0	0	0	0
41	G.G.	2	3	2	0	0	0	0
42	W.G.	2	4	3	2	0	0	0
43	C.G.	2	4	3	3	0	0	0
44	C.McE.	1	4	2	2	0	0	0
45	J.L.	3	3	2	3	0	0	0
46	A.N.	2	3	3	0	0	0	0
47	A.O.	3	2	3	0	2	2	0
48	G.M.	2	3	4	0	3	2	2
49	E.McA.	3	2	3	3	3	0	0
50	E.D.	2	2	0	0	0	0	0
51	A.T.	2	2	0	0	0	0	0
52	C.McD.	3	2	0	0	0	0	0
53	J.G.	3	2	0	0	0	0	0
54	E.O.	3	3	0	0	3	0	0

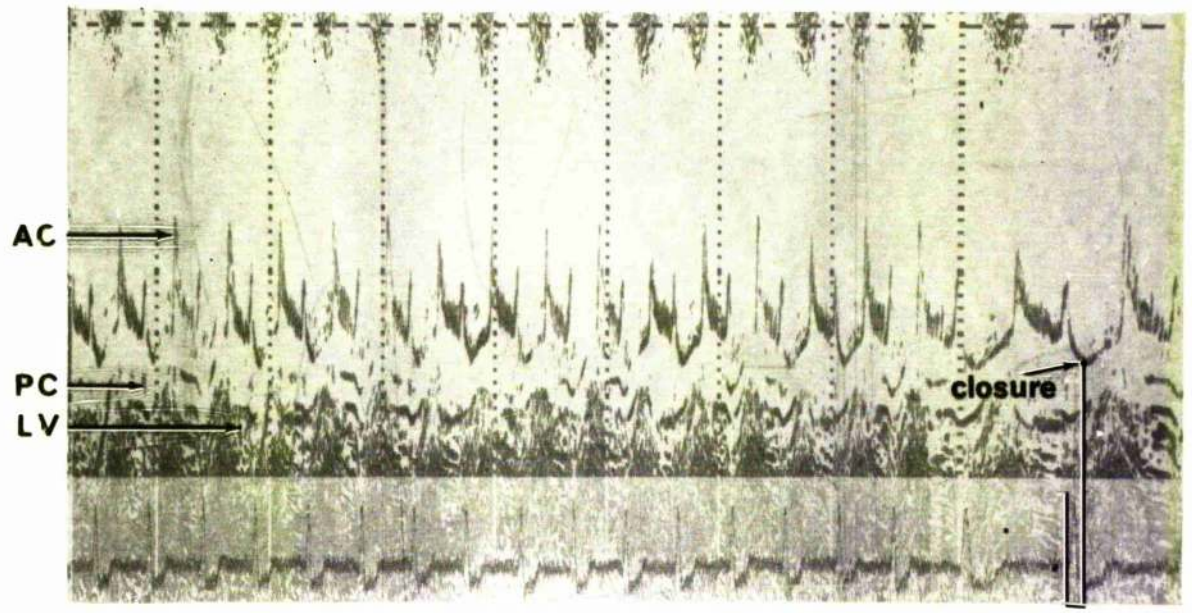


FIG. 6,1 METHOD USED TO TIME MITRAL SYSTOLIC CLOSURE

TABLE 6,5 MITRAL DCR IN AORTIC VALVE DISEASE

	DCR mm/sec.	AR	AS	
43	350	4	2	
1	338	4	0	
37	330	4	1	
21	280	4	2	
51	266	2	2	
7	255	4	0	
31	233	3	2	DCR Normal Range
2	232	4	0	
44	219	4	1	
50	210	2	2	
33	200	3	2	
32	196	3	2	
17	190	0	4	
39	170	2	2	
16	170	0	3	
27	160	3	4	
52	125	2	3	
35	80	2	3	
46	75	3	2	
40	43	2	4	
22	35	3	3	

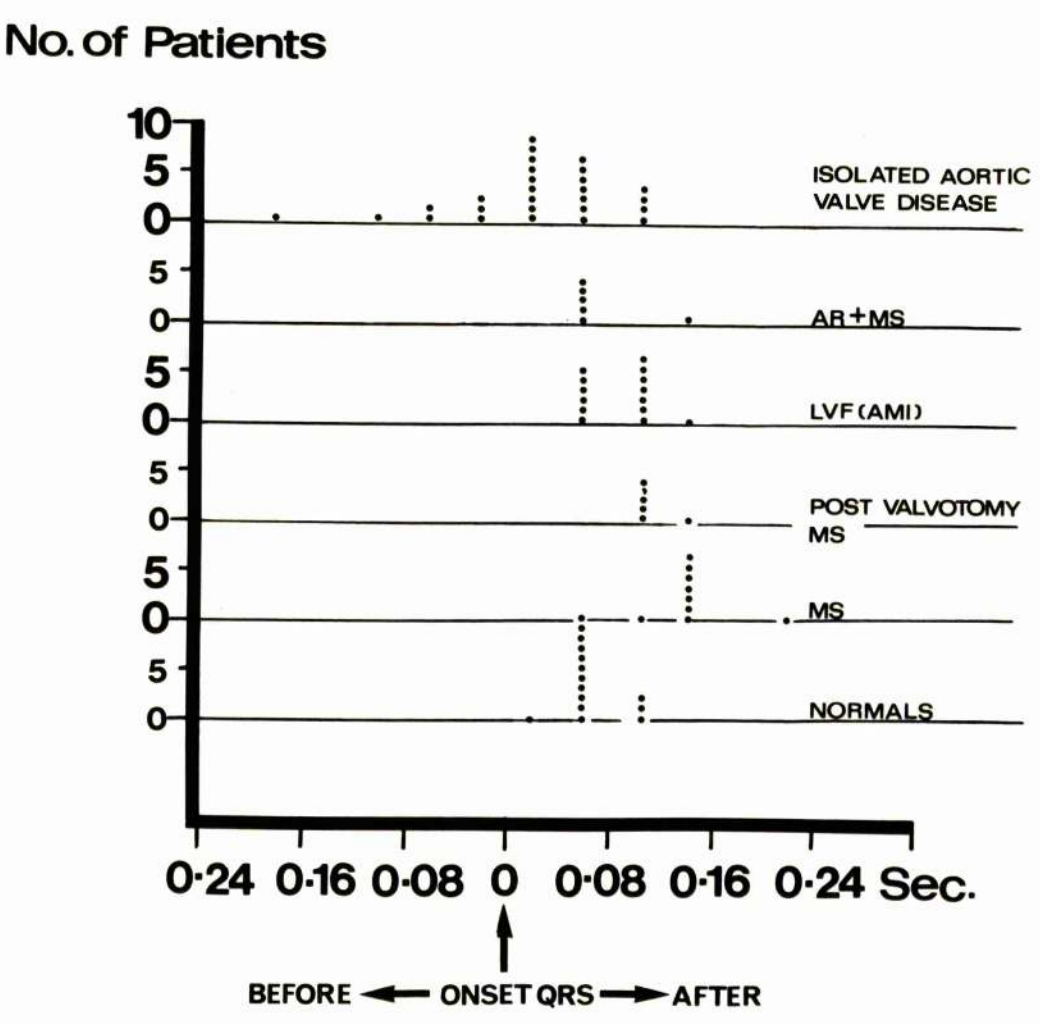
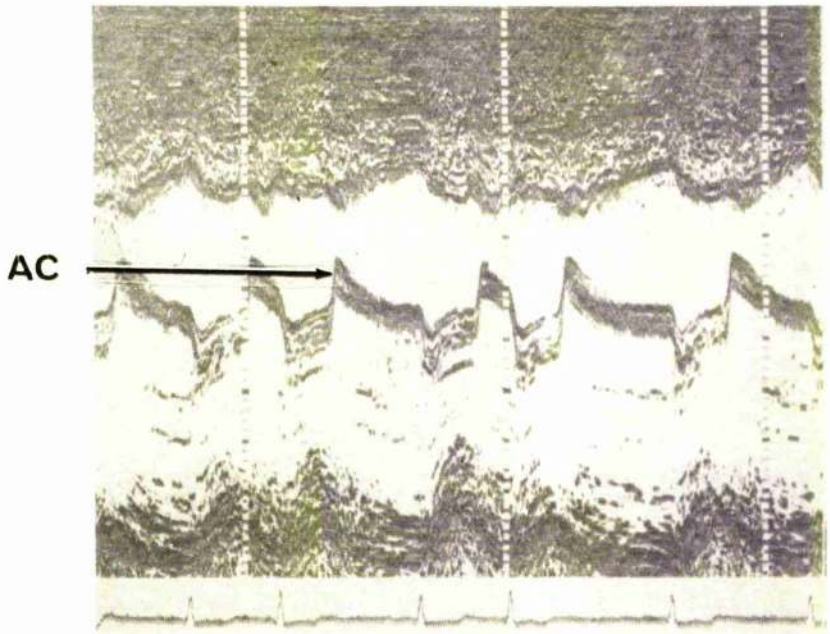
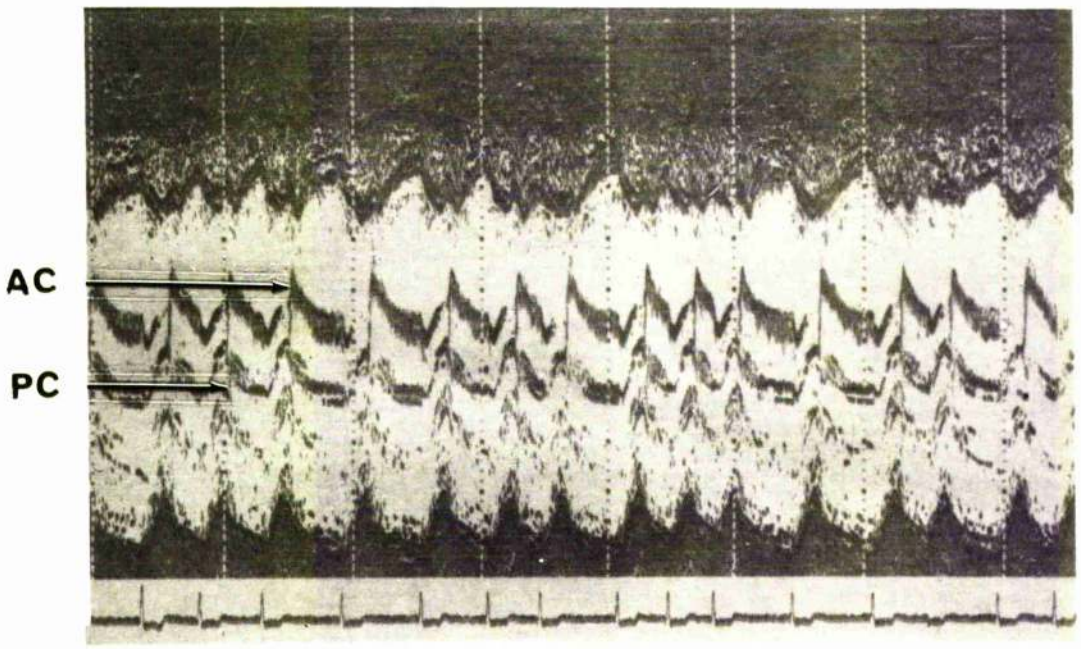


FIG. 6,2 MITRAL SYSTOLIC CLOSURE TIMES



(a)



(b)

FIG. 6,3 - See Text

CHAPTER 7

ECHOCARDIOGRAPHY IN ISCHAEMIC MITRAL INCOMPETENCE

The clinical features of non-rheumatic subvalvar mitral incompetence are well established (Raftery, Oakley and Goodwin, 1966; Caves, Sutton and Paneth, 1973). It is most commonly idiopathic, post-infective or ischaemic (Caves et al., 1973). It is possible that the ischaemic mitral regurgitation associated with acute myocardial infarction and that associated with previous infarction have different mechanisms. Thus, in acute myocardial infarction, papillary muscle rupture (Sanders, Neuburger and Ravin, 1957; Caves et al., 1973), papillary muscle infarction (Raftery et al., 1966) and papillary muscle ischaemia (Burch, De Pasquale and Phillips, 1968) produce varying degrees of mitral regurgitation. In contrast, in patients presenting with previous infarction and subvalvar incompetence, there is little evidence of papillary muscle fibrosis: instead, the chordae tendineae to the mitral anterior cusp are stretched or ruptured and there is consequent cusp prolapse (Caves et al., 1973).

The precise mechanisms of subvalvar mitral regurgitation have been the subject of much speculation (Burch, De Pasquale and Phillips, 1963; Caves et al., 1973) but remain uncertain: angiography has been unhelpful. Mitral valve echocardiography might reasonably be expected to elucidate the problem. Tallury, De Pasquale and Burch (1972) have measured the mitral diastolic closure rate in acute papillary muscle dysfunction and Cohn and his co-workers have described the echocardiogram in both chordal rupture (Sweatman et al., 1972) and papillary muscle dysfunction (Burgess et al., 1973). However, these investigations have failed to define echocardiographic features diagnostic of subvalvar mitral regurgitation and have provided little new information on its mechanics.

The/...

The aim of the present study was to review the echocardiographic findings in a group of patients with ischaemic mitral incompetence.

Patients

8 patients were studied: the clinical features and the electrocardiographic and radiographic findings are summarised in Table 7,1. None had a history of rheumatic fever and all had Q wave evidence of myocardial infarction. None of the patients underwent haemodynamic or angiographic investigation. All except Case 1 have died: post-mortem examinations were carried out in Cases 2 and 7.

Case 1 developed signs of mitral regurgitation and cardiac decompensation three days after inferior myocardial infarction. Left heart failure responded to standard medical treatment and at follow-up 6 months later, the murmur had diminished in intensity.

Case 2 who was on anti-failure therapy following a previous myocardial infarction, developed gross pulmonary oedema on the second day of an acute inferior infarct and died one day later. No murmur was detected but both recent and old infarcts involving the posterior papillary muscle were demonstrated at post-mortem.

Cases 3 to 8 presented with auscultatory evidence of mitral regurgitation: phonocardiograms were recorded in Cases 4 (Fig. 7,2), 5, 6 and 7 and in each instance demonstrated a pansystolic diamond-shaped murmur at the mitral area. 4 patients were in sinus rhythm and 3 had electrocardiographic evidence of left atrial hypertrophy. All were decompensated and all had at least moderate cardiac enlargement. Fluoroscopy detected a calcified aneurysm of the antero-lateral aspect of the left ventricle in Case 8 (Fig. 7,3) and hypokinetic areas at the same site in Cases 3 and 6. Post-mortem examination in Case 7 demonstrated a shrunken, fibrotic posterior papillary muscle and marked left atrial dilatation.

Methods

Mitral echograms were recorded in all patients. In addition, satisfactory/...

satisfactory simultaneous records from the interventricular septum and the left ventricular posterior wall were obtained in 5 cases. The equipment, technique and recording methods were as previously described (Chaps. 1 and 2). Measurements were made as follows:- the mitral diastolic closure rate as detailed in Chap. 2, the total amplitude of the mitral echogram by the method of Edler (1967), the diastolic excursion of the anterior cusp as described in Chap. 3 and left ventricular dimensions, volumes and ejection fraction by the methods of Pombo, Troy and Russell (1971), with the minor modification that the shortest distance between the endocardial surfaces of the interventricular septum and the left ventricular posterior wall was used as a measure of end-systolic dimension.

Results

The results have been tabulated (Table 7,2).

I. The mitral valve

Amplitude

The mean total amplitude of the mitral echogram was 22.4 $\pm$ 3.4 mm. This is significantly lower ($P<0.001$) than the mean value (29.1 $\pm$ 4.3 mm) observed in a series of normal subjects (Chap. 8). The mean diastolic excursion of the anterior cusp, 18.7 $\pm$ 2.6 mm was also significantly ($P<0.001$) below normal (Chap. 8).

Diastolic closure rate

The mean mitral diastolic closure rate was 317 $\pm$ 50 mm/sec. This is significantly higher ($P<0.001$) than the mean value (176 $\pm$ 29 mm/sec) observed in a series of normal subjects (Chap 2)

Systolic cusp apposition

Normal systolic apposition of the anterior and posterior mitral leaflets was demonstrated in none of the patients (Figs. 7,1 to 7,3). In 4 instances, the relative positions of the cusps appeared to reverse during systole. Thus, in systole, the posterior cusp echogram apparently arched forwards and was recorded in front of the anterior cusp echogram which was flat/...

flat or sagged posteriorly (Fig. 7,1, Case 3 and Fig. 7,2).

Diastolic oscillation

A rapid, fine, diastolic oscillation of the mitral anterior and posterior cusp echograms was observed in all the patients. The oscillation was obvious on both the A and TP scans: it is particularly well seen in the tracings from patients 2, 4 and 8 (Figs. 7,1; 7,2; and 7,3).

II. The left ventricle

Ventricular volumes and the ejection fraction

Comparing the results with those from a series of normal subjects (Chap. 8), end-diastolic volume was increased in 4 patients while ejection fraction was severely reduced and end-systolic volume was increased in all 5.

Interventricular septum

In cases 5, 6 and 8, maximal posterior displacement of the interventricular septum occurred in early diastole i.e. septal motion was reversed.

Discussion

In a small series of cases of post-infarction mitral regurgitation (Caves and Paneth, 1973; Caves et al., 1973), impairment of left ventricular function was invariable and left ventricular aneurysm was common. The usual operative findings were stretching or rupture of the anterior cusp chordae: there was little evidence of papillary muscle fibrosis. These observations tend to support the theories of Burch et al. (1963 and 1968) on the causal role of both left ventricular dilatation and left ventricular aneurysm in subvalvar mitral incompetence but suggest that the same workers may have over-estimated the importance of papillary muscle fibrosis in the context of previous infarction. There is thus evidence that the mitral regurgitation associated with previous myocardial infarction is anatomically and functionally distinct from other types of subvalvar incompetence.

Angiography and pressure measurements were not feasible in the present study/...

study. The diagnosis of ischaemic subvalvar mitral incompetence was thus of necessity clinical and was based on unequivocal electrocardiographic evidence of myocardial infarction and, with the exception of Case 8 in whom the mitral murmur was variable, on classical signs of mitral regurgitation. Mitral regurgitation was presumed to be due to acute papillary muscle ischaemia or infarction in Case 1 and to papillary muscle fibrosis in the cases examined post-mortem. Left ventricular dilatation and left ventricular dyskinesis, which was demonstrated in three instances, were thought to be possible causal factors in the remaining patients.

In view of the findings on the plain chest x-ray and at fluoroscopy, the increased left ventricular dimensions demonstrated by ultrasound were obviously to be expected. The consistently low ejection fractions are in line with angiographic observations in post-infarction mitral incompetence (Caves et al., 1973). It should, however, be noted that septal motion was abnormal in 3 instances and that the reduction in ejection fraction may therefore have been over-estimated. Two of these cases had antero-septal infarcts but the third had no electrocardiographic abnormality to explain the reversal of septal movement.

Mitral echogram amplitude and left ventricular volumes are negatively correlated (Chap. 8): abnormally low values were recorded in the present study when the cardio-thoracic ratio exceeded 64%. Burch et al. (1968) have postulated that in the presence of left ventricular dilatation, contraction of the papillary muscles may produce downward traction on the mitral cusps in systole. It has been shown in the canine heart that the subvalvar apparatus restrains the mitral leaflets during both diastole and systole (Padula, Cowan and Camishion, 1968). Hence, it is suggested that chordal stretching with consequent restriction of diastolic cusp excursion may occur as a dilated left ventricle fills. These theories could explain both the low total echogram amplitude and the low diastolic excursion of the anterior cusp observed here and in other studies (Burgess et al., 1973; Millward, McLaurin and Craige, 1973).

While/...

While diastolic oscillation of the mitral cusps is a constant feature of ischaemic mitral incompetence, it has also been observed in subvalvar regurgitation of different aetiology (Fig. 8, 8). The oscillation is more rapid than is usual in aortic regurgitation and is so striking that it is surprising it has not been described previously. No firm conclusions can be reached on its cause; it may simply be due to increased mitral flow or may reflect abnormal tethering of the cusps. Theoretically, if the stretched chordae become taut in diastole, the increased tension on the cusps could cause them to oscillate as the ventricle fills. Alternatively, the oscillation could result from failure of lax chordae to restrain the leaflets during diastole. The first explanation is favoured by the low amplitude and high frequency of the oscillation and by the reduction in diastolic excursion of the leaflets.

In Case 8, the systolic closure movement of the anterior cusp was interrupted (Fig. 7,3). The same abnormality was observed frequently in hypertrophic obstructive cardiomyopathy (Fig. 7,4a), less often in congestive cardiomyopathy (Fig. 7,4b) and once in the acute infarct series (Fig. 7,4c and Chap. 3). Feigenbaum and his co-workers have attributed the anomaly to marked elevation of the left ventricular end-diastolic pressure (Konecke et al., 1973): the pressures in the patients with cardiomyopathy (Fig. 7,4a and b) are in line with this interpretation. It is of interest that the infarct patient in whom systolic closure was interrupted had a normal mitral diastolic closure rate despite radiographic evidence of a high left atrial pressure and was thought on this basis to have a non-compliant left ventricle (Chap. 3).

The diastolic closure rates in this study are higher than those observed by Burgess et al. (1973) in ischaemic mitral incompetence and may reflect more severe reflux or more normal ventricular compliance in the present series. It should, however, be noted that in the presence of diastolic oscillation and prominent chordal echoes, particular care is necessary to ensure that a mitral echogram/...

echogram of submaximal amplitude and with an artefactually low diastolic closure rate is not recorded.

A definitive echocardiographic diagnosis of non-rheumatic mitral regurgitation can be made only when failure of systolic apposition of the mitral leaflets is demonstrated. Unfortunately, the systolic phase of the mitral echogram may be difficult to interpret (Figs. 7,1 to 7,4). The problem arises because multiple echoes are often recorded: they are particularly prominent when the left ventricle is dilated. It is postulated that the additional echoes originate from the chordae tendineae. The chordae insert at, and up to 1 cm. behind, the free margins of the ventricular surface of the mitral cusps (Lam et al., 1970). Thus, when the ultrasonic beam is directed in systole through the free margins of the leaflets, it could theoretically traverse the chordae inserting beyond them as in Fig. 7,5.

Fig. 7,6 is from a patient with chronic renal failure, congestive cardiac failure and a variable, soft apical systolic murmur (Case 15, Chap. 9). In complexes 1 and 2 there are multiple systolic echoes: it is suggested that the most anterior echoes originate from the anterior cusp chordae and that behind them in sequence are echoes from the posterior cusp, the anterior cusp and the chordae to the posterior cusp. In complex 4, no posterior cusp echogram has been recorded in diastole and only two echoes have been recorded in systole. Comparing the waveform of these two systolic echoes with those in complexes 1 and 2, it is suggested that they derive not from the anterior and posterior cusps but from the anterior cusp and its chordae. There is evidence that Burgess et al. (1973) and Millward et al. (1973) have failed to appreciate this point and have accordingly misdiagnosed wide systolic separation of the mitral leaflets on similar echograms.

Anterior cusp prolapse was suspected in Cases 1 and 2 (Fig. 7,1) because it was particularly difficult to record a continuous echogram from that structure during systole. A continuous sweep from the aortic root to the anterior cusp would have provided more definitive information but the validity of the manoeuvre was/...

was not appreciated at the time the records were made. In four instances, including cases 3 and 4 (Figs. 7,1 and 7,2), the positions of the mitral leaflets appeared to reverse in systole: the same phenomenon is seen in Fig. 7,6. The finding could have been easily explained had the ultrasonic beam passed through the leaflets to the left atrial instead of the left ventricular wall; in these circumstances echoes from a prolapsed anterior cusp might be picked up within the atrium, intervening between the posterior cusp and left atrial wall echoes. However, the beam in fact traversed the left ventricular wall and Fig. 7,7 offers a tentative explanation for the appearances recorded. The posterior cusp chordae function normally but the anterior cusp chordae are slack. Thus, during systole the posterior cusp is dragged downwards by the subvalvar apparatus of the dilated ventricle and as a result the poorly tethered anterior cusp moves above it and sags towards the left atrium. The free edge of the anterior cusp is thus recorded between the posterior cusp and the left ventricular wall.

It is interesting that the echocardiographic findings in the present study are essentially similar to those reported by Millward et al. (1973) in congestive cardiomyopathy with mitral regurgitation. Both disorders are associated with ventricular dilatation and impaired ventricular function. It is therefore possible to speculate that these abnormalities, rather than specific defects of the papillary muscles or the chordae, are the basic cause of ischaemic mitral incompetence. Burch et al. (1968) have indicated how ventricular dilatation and ventricular dyskinesis could cause mitral incompetence: the present investigation tends to support their theories. Thus, in the patients studied, because the amplitude of the echogram was never increased and systolic separation of the leaflets was rarely gross, mitral regurgitation appeared to be produced by abnormal systolic positioning of the leaflets within the ventricle rather than by cusp prolapse.

Millward et al. (1973) studied patients with congestive cardiomyopathy complicated by mitral incompetence. They concluded that echocardiography can be used to diagnose congestive cardiomyopathy. This is clearly not so: indeed, in/...

in my own experience, ultrasound cannot differentiate congestive cardiomyopathy without mitral reflux from ischaemic mitral incompetence.

Summary

Mitral echograms and the ultrasonic dimensions of the left ventricle were recorded in 8 patients with ischaemic mitral incompetence: the results were compared with those from normal subjects. The findings were as follows:- 1) Mitral echogram amplitude was reduced ($P<0.001$). 2) Mitral diastolic closure rate was increased ($P<0.001$). 3) The mitral cusp oscillated in diastole. 4) Systolic apposition of the cusps was consistently abnormal. 5) Left ventricular ejection fraction was grossly reduced.

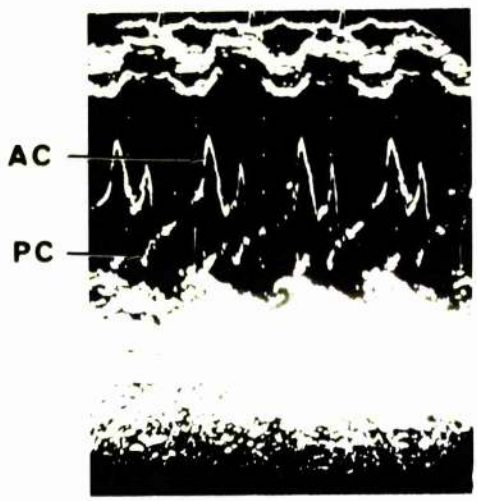
It is concluded that ischaemic mitral incompetence and congestive cardiomyopathy cannot be distinguished echocardiographically. It is suggested that abnormal positioning of the leaflets within the left ventricle, rather than cusp prolapse, is the usual mechanism of ischaemic mitral incompetence and that the low echogram amplitude associated with this disorder is due to chordal stretching in diastole.

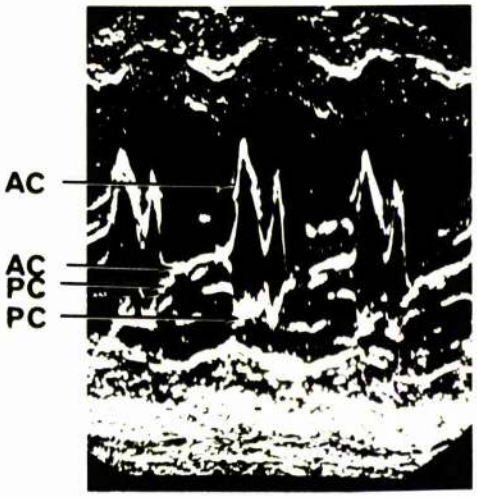
TABLE 7,1 CLINICAL FEATURES

	Time from infarction	systolic murmur	Rhythm	Electrocardiogram		Chest radiograph	
				LA hypertrophy	Infarct. site	CTR %	LVF
1	3 days	+	Sinus	-	Inferior	60	+
2	2 days & 6 months	-	Sinus	-	Inferior	57	++
3	3 months	+	Sinus	+	Antero-lateral & inferior	65	++
4	2 years	+	Sinus	±	Inferior	66	+
5	2 years	+	Sinus	+	Anterior & Inferior	60	+
6	4 years	+	A.F.		Anterior & Inferior	66	++
7	2 years	+	A.F.		Anterior & Inferior	60	++
8	8 years	variable	Sinus	+	Anterior	69	+

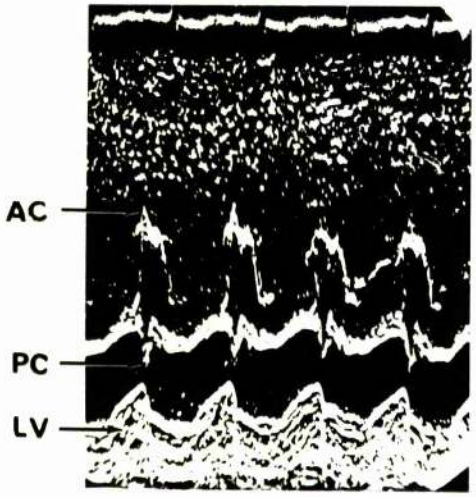
TABLE 7,2 ECHOCARDIOGRAPHIC FINDINGS

	Mitral anterior cusp echogram				End-diastolic volume ml.	End-systolic volume ml.	Stroke volume ml.	Ejection fraction
	Total amplitude mm.	DCR MM/sec	Diastolic oscillation	Systolic cusp reversal				
1	27	310	+		334	180	154	0·46
2	25	360	+					
3	19·5	340	+	+				
4	21·5	400	+	+	251	140	111	0·44
5	25	290	+	+	143	89	54	0·38
6	23	320	+		228	146	82	0·36
7	21·5	266	+	+				
8	16·5	250	+		455	233	222	0·49
Mean ± 1 SD	22·4 ± 3·4	317 ± 50						0·43 ± 0·06

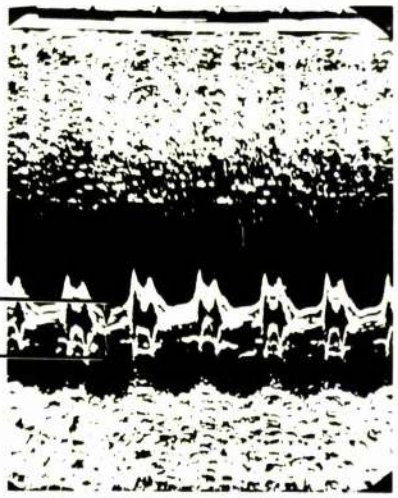




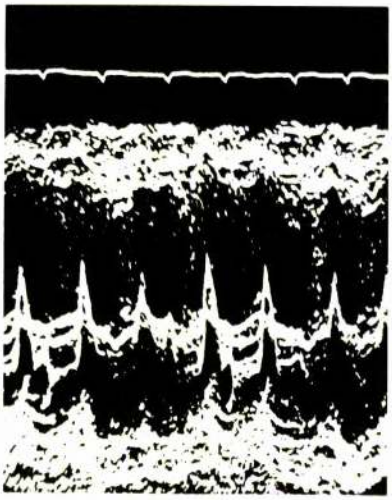
a) CASE 1



b) CASE 2

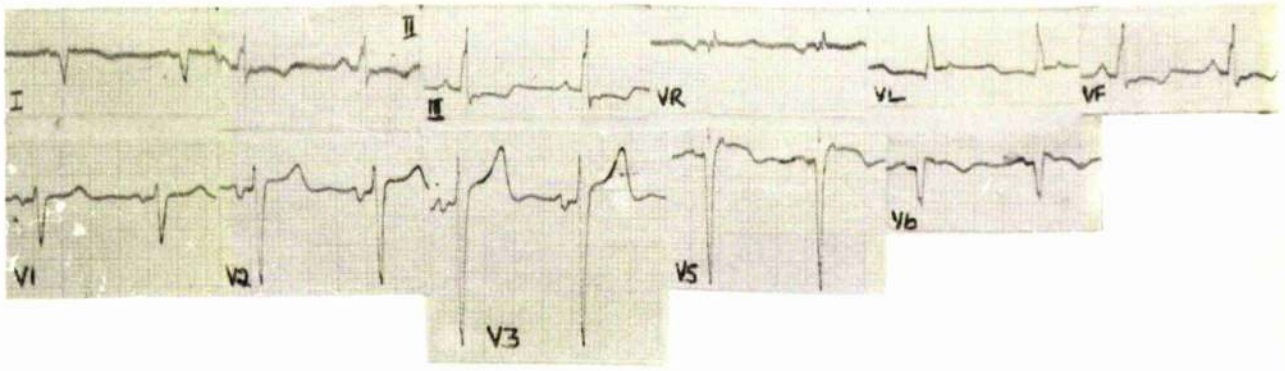


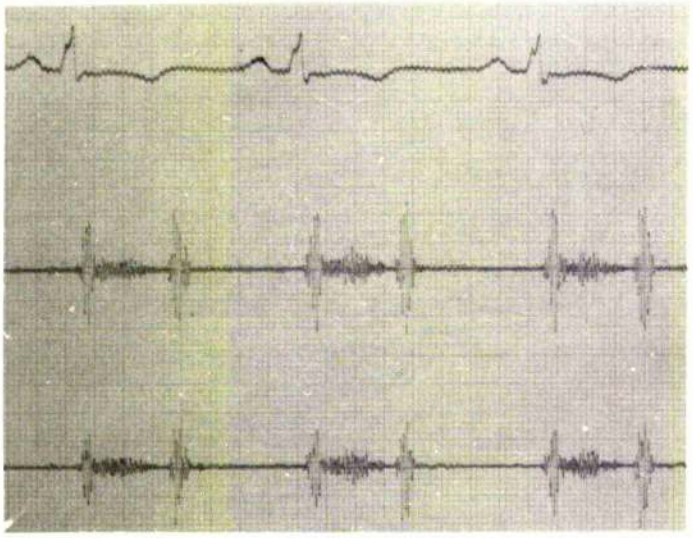
c) CASE 3



d) CASE 6

FIG. 7,1





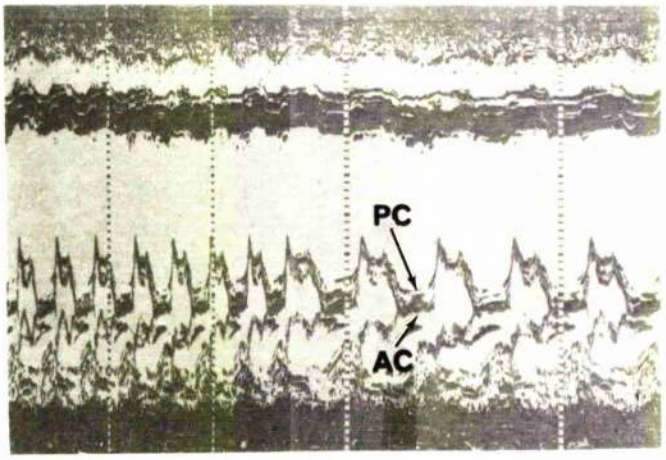
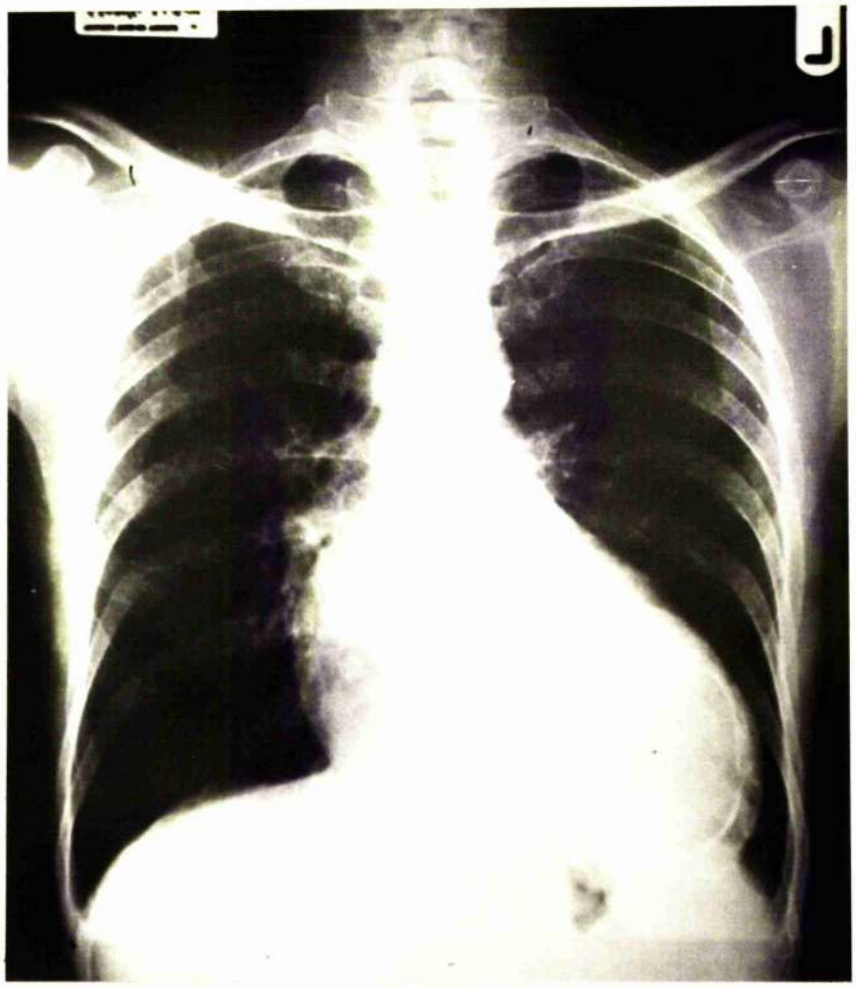


FIG. 7,2

CASE 4 - ECG, PHONOCARDIOGRAM AND ECHOCARDIOGRAM



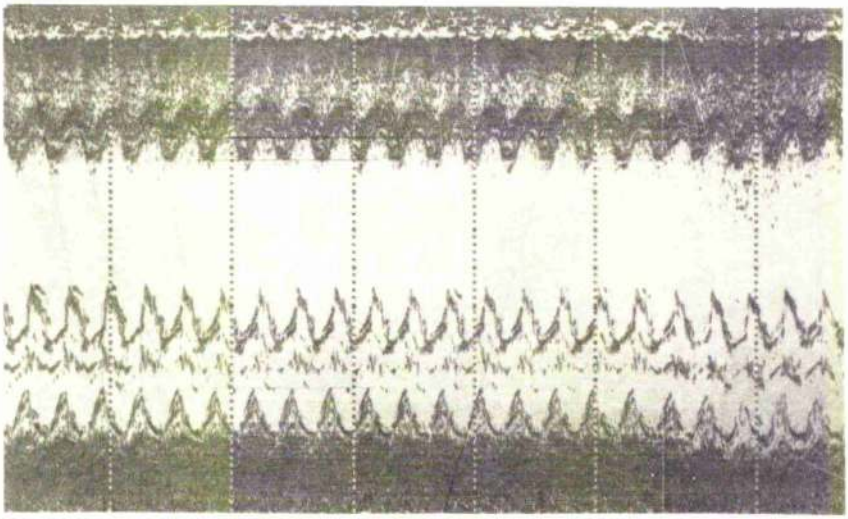
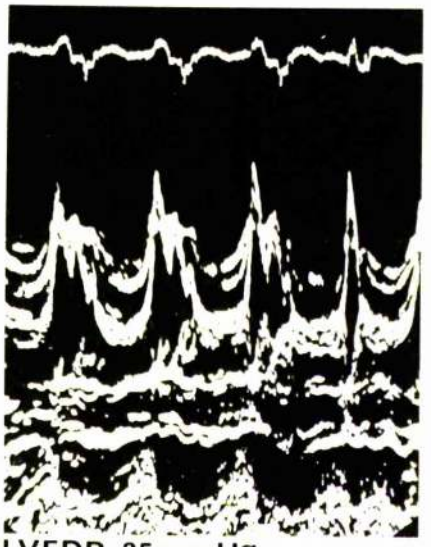
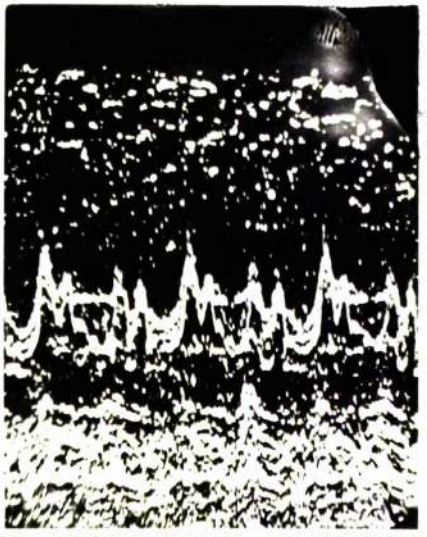


FIG. 7,3 CASE 8 - ECHOCARDIOGRAM AND CHEST X-RAY WITH CALCIFIED VENTRICULAR ANEURYSM.

a) HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY



b) CONGESTIVE CARDIOMYOPATHY



c) ACUTE MYOCARDIAL INFARCTION

FIG. 7,4 INTERRUPTED MITRAL SYSTOLIC CLOSURE

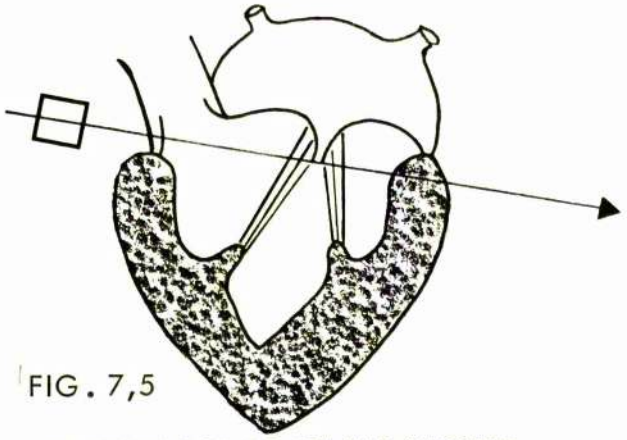


FIG. 7,5

AN ULTRASONIC BEAM DIRECTED THROUGH THE FREE MARGINS OF THE MITRAL LEAFLETS WILL TRAVERSE THE CHORDAE INSERTED BEYOND THEM.

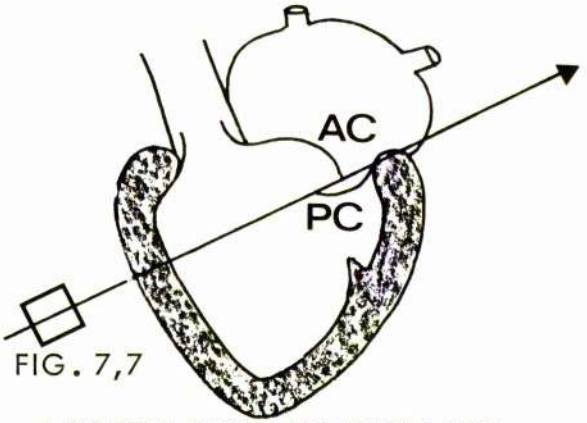


FIG. 7,7

A POSSIBLE EXPLANATION FOR CUSP "REVERSAL" IN SYSTOLE.

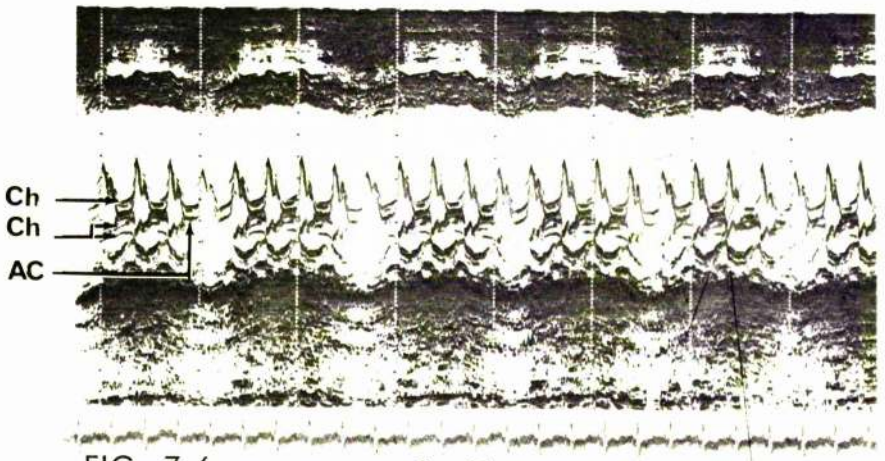


FIG. 7,6

See Text

CHAPTER 8

THE DIMENSIONS, VOLUMES AND PERFORMANCE OF THE LEFT VENTRICLE

The potential of cardiac ultrasound in the assessment of the left ventricle is becoming clear but is as yet largely unexploited. Because measurements can be repeated, because patients unfit for invasive investigation can be studied and particularly because ventricular performance is unaffected by the procedure, it is to be expected that echocardiography of the left ventricle will be used widely in adult cardiology and in clinical pharmacology. In this thesis, the results of echocardiographic studies of the left ventricle in chronic renal failure (Chap. 9) and ischaemic heart disease (Chaps. 7 and 8) have been reported. Although parallel angiographic and haemodynamic data were not obtained, the results are thought to be valid because my own observations, reported below, and the findings of workers who have had the opportunity to compare echocardiographic and angiographic assessments, indicate that the ultrasonic method is acceptably accurate in a variety of cardiac disorders.

Ultrasonic assessment of the left ventricle - a review of the literature

The identification of the echoes from the interventricular septum led to the echocardiographic estimation of left ventricular dimensions (Popp et al., 1969). The ventricular dimension which is measured by ultrasound has been shown to approximate the angiographic mean minor axis and to correlate significantly with the semilength of the angiographic major axis (Pombo, Troy and Russell, 1971; Gibson, 1973). The cube of the ultrasonic dimension can be taken as a measure of left ventricular volume because the left ventricular cavity can be represented by an ellipsoid and its shape is uniform from patient to patient (Dodge and Baxley, 1969). Ventricular volumes derived by this method agree closely/...

closely with estimations made from biplane angiograms (Pombo et al., 1971; Gibson, 1973). When the left ventricle dilates its cavity can no longer be represented by an ellipsoid. Nonetheless, the cube of the ultrasonic dimension and angiographic estimates of ventricular volume remain significantly correlated (Ludbrook et al., 1973).

Gibson (1973) has suggested that echocardiographic estimates of stroke volume are less likely to be affected by abnormalities of left ventricular shape than are single measurements of end-diastolic and end-systolic volume. In practice, left ventricular stroke volume determined by ultrasound has been found to correlate well with measurements made by the dye-dilution (Pombo et al., 1971) and the Fick methods (Popp and Harrison, 1970).

Cooper et al. (1972) found close agreement between ultrasound and cineangiographic estimates of ejection fraction. They observed that, in the dilated ventricle, more accurate results were obtained by cubing the ultrasonic dimension than by using the regression equation of Fortuin et al. (1971) for ventricular volume. They have subsequently shown that the echocardiographic estimation of ejection fraction is in general accurate in patients with abnormalities of left ventricular wall motion (Ludbrook et al., 1973).

The left ventricular cavity can be outlined by multidimensional echocardiography and area-length methods (Greene et al., 1967) can thus be used to measure ventricular volumes on multiscans (Roelandt et al., 1974). Provided recording methods improve, it seems likely that multiscanning will replace angiography in the assessment of the dimensions and performance of the left ventricle.

Before the investigation reported in Chap. 9 was undertaken, the dimensions, volumes and ejection fraction of the left ventricle were measured by ultrasound in a group of normal subjects and in patients with various cardiac disorders. It was hoped that by comparing the results within and between the groups, it would be possible to decide whether values of sufficient accuracy for practical purposes could be derived from the dyskinetic, the dilated and the decompensated ventricle. However, the main aim of this preliminary study was to identify problems involved in recording and measuring the ultrasonic dimensions.

Subjects

58 subjects were studied. They have been grouped as follows:-

Group 1

13 subjects judged to have normal hearts. Their ages ranged from 14 to 54 (mean 30) years and 9 were female. All were hospital in-patients who were either being treated for a dermatological disorder or were awaiting a minor gynaecological procedure.

Group 2

8 subjects with isolated and at least moderately severe mitral stenosis. All were awaiting closed mitral valvotomy and 6 had been catheterised.

Group 3

8 subjects who had undergone successful mitral valvotomy between 3 and 6 months previously. None had clinical signs of mitral regurgitation or significant aortic valve disease.

Group 4

4 subjects with isolated rheumatic mitral valve disease in whom significant mitral regurgitation had been demonstrated angiographically.

Group 5

7 subjects judged clinically to have ischaemic mitral incompetence. All had a mitral diastolic closure rate above 200 mm/sec., an apical pansystolic murmur, Q wave evidence of myocardial infarction and radiographic signs of decompensation/...

decompensation.

Group 6

3 subjects with non-rheumatic mitral regurgitation which was considered not to have an ischaemic basis. One patient developed severe mitral regurgitation in the course of a subacute bacterial endocarditis. The other two, who were known to have systemic hypertension presented with acute mitral incompetence. In each case the mitral diastolic closure rate was above 300 mm/sec.

Group 7

3 subjects with predominant aortic stenosis in whom a significant aortic systolic gradient had been demonstrated.

Group 8

12 subjects with moderate or severe aortic regurgitation who were awaiting aortic valve replacement. Left ventricular end-diastolic pressure had been measured in 8 cases and was elevated in 5 of them.

Methods

The equipment has been described (Chaps. 1 and 2). The examination was made with the patients supine. The transducer was positioned to record echoes from the mitral leaflets within the left ventricle. It was then angled downwards to display simultaneous echograms from the endocardial surfaces of the interventricular septum and the left ventricular posterior wall.

When septal motion was reversed, the shortest distance between the septal and posterior wall echoes was taken as a measure of end-systolic dimension: otherwise, dimensions were measured as in Fig. 8,1. Volumes were derived by cubing the dimensions and ejection fraction was calculated by the method of Pombo et al., (1971).

Results

Measurement problems were encountered when posterior wall excursion varied (Fig. 8,2), when the origin of the posterior echo was uncertain (Fig. 8,3) and when/...

when septal motion was reversed (Fig. 8,4).

The results have been tabulated (Tables 8,1 and 8,2).

End-diastolic dimension and end-diastolic volume

The mean values for end-diastolic dimension and end-diastolic volume in the normal subjects were 4.7 ± 0.5 cm and 109 ± 31 ml respectively. Significant increases were observed in aortic regurgitation and pure mitral regurgitation but the findings in rheumatic mitral incompetence did not reach statistical significance ($P<0.10$). The group with mitral stenosis did not differ from normal.

End-systolic dimension and end-systolic volume.

The mean values for end-systolic dimension and end-systolic volume in the normal subjects were 2.8 ± 0.5 cm and 23 ± 12 ml respectively. Significant increases were observed in aortic regurgitation, pure mitral regurgitation and the post-valvotomy group. The findings in mitral stenosis and rheumatic mitral incompetence ($P<0.10$) did not differ from normal.

Stroke volume and cardiac output

The mean values for left ventricular stroke volume and cardiac output in the normal subjects were 83 ± 23 ml and 6.5 ± 2.2 l respectively. Although higher values were also found in rheumatic and ischaemic mitral incompetence, significant increases were observed only in aortic regurgitation and ischaemic mitral regurgitation.

Ejection fractions

Ejection fraction in the normal group was 0.8 ± 0.065. Significantly lower values were found in ischaemic ($P<0.001$) and non-ischaemic ($P<0.05$) mitral regurgitation, aortic regurgitation ($P<0.05$) and in the post-valvotomy group ($P<0.001$).

Discussion

Inaccuracies are inevitable in the measurement of left ventricular volume and/...

and ejection fraction by single plane echocardiography. Thus, the basic assumptions about the shape of the cavity and the uniformity of wall motion may be invalid in individual patients, while possible errors due to shortening of the long axis and to rotation and lateral movement of the heart can neither be identified nor compensated for.

Some errors can be avoided. Dimensions should be measured over a series of cardiac cycles as although beat to beat variations are usually small, they may on occasion be significant (Fig. 8,2): in the example shown estimates of end-diastolic volume and ejection fraction varied from 136 to 216 ml and from 0.41 to 0.63 respectively. Because the ultrasonic dimension decreases at the level of the posterior papillary muscle (Chap. 4), measurements must be made above that structure and immediately below the mitral valve. In recording Fig. 8,3, the ultrasonic beam was swept downwards from the mitral valve towards the apex of the ventricle: the changes in dimension are obvious.

Although the method used here to measure end-systolic dimension in the presence of abnormal septal movement may be empirical, there is no evidence that it is seriously inaccurate. Thus, ejection fraction derived by this method is normal in atrial septal defect (Fig. 8,4) and, in the present series, was reduced in ischaemic mitral incompetence.

In that left ventricular end-diastolic, end-systolic and stroke volumes were normal in mitral and aortic stenosis and significantly increased in mitral and aortic regurgitation, the findings of this study suggest that even in the presence of left ventricular dilatation, the cube of the ultrasonic dimension provides a useful estimate of ventricular volume. It is not possible to comment on the absolute accuracy of the results. However, it is interesting to note that very similar results, both in numerical terms and in terms of the significance of the differences between patients and normals were obtained by Gibson and Brown (1973) in an echocardiographic study of normal subjects and of patients with valvular disease.

The ejection fractions in this series of normal subjects were higher than those/...

those reported by Gibson and Brown (1973) and did not approach the lower limit of normal set by Kennedy et al. (1966) from angiographic data. Because until recently it could only be determined angiographically, there is little published information on the ejection fraction of the normal heart: an echocardiographic study of a large number of normal subjects would be valuable. In the present investigation, although significant reductions were observed in other groups, ejection fraction was, as might be expected, lowest in ischaemic mitral incompetence. Heller and Carleton (1970) and Holzer et al (1973) have demonstrated impaired left ventricular performance in some patients with mitral stenosis: the reduced ejection fraction in one patient with mixed mitral valve disease and the low mean value in the post-valvotomy group are in line with their findings.

Diastolic velocity of the left ventricular posterior wall

Left ventricular filling has been studied by various methods: an early diastolic phase of rapid filling and a subsequent phase of reduced inflow have been demonstrated (Nolan et al., 1969; Dodge and Baxley, 1969; Kumar and Spodick, 1970; Rushmer, 1972). Fogelman et al. (1972) have used ultrasound to investigate the diastolic motion of the posterior wall of the normal and the ischaemic left ventricle.

The aim of this study was to evaluate the posterior wall diastolic velocity as an index of left ventricular filling by comparing the results from normal subjects and patients with valvular disease and by examining the relation between the mitral diastolic closure rate and the posterior wall diastolic velocity.

Subjects and Methods

The ultrasonic tracings from the 58 subjects of the previous study (Chap. 8), from 25 patients on regular dialysis therapy (Chap. 9) and from 4 additional normal subjects were reviewed. Those on which the posterior wall diastolic

velocity/...

velocity could be measured were selected and form the basis of the present study.

Left ventricular posterior wall diastolic velocity was measured as the slope of the line joining the most anterior point on the echogram with the point where rapid posterior displacement ends (Fig. 8,1).

The results from 17 normal subjects and 40 patients with valve lesions were compared (Table 8,2). The relation between the mitral diastolic closure rate and posterior wall diastolic velocity was examined in 10 normal subjects, 8 dialysis patients and 11 patients with valvular disorders (Appendix).

Results

Posterior wall diastolic velocity in normal subjects and patients with valvular disease

The results have been tabulated (Table 8,2).

In the normal group, posterior wall diastolic velocity ranged from 76 to 156 mm/sec with a mean of 114 $\pm$ 28 mm/sec: with the exception of the pre-valvotomy group, some patients in each group had values within the normal range. Posterior wall diastolic velocity was significantly reduced in mitral stenosis and mixed mitral valve disease. No other consistent trends were observed; in aortic regurgitation, posterior wall diastolic velocity was increased in 2 and normal in 8 cases while in mitral regurgitation it was increased in one, decreased in one and normal in 3 cases.

Posterior wall diastolic velocity was positively and significantly correlated with stroke volume ($r = 0.41$; $P<0.02$): when patients with rheumatic mitral valve disease were excluded, the correlation was no longer significant ($r = 0.27$; $P>0.10$). Posterior wall diastolic velocity did not correlate significantly with ejection fraction.

Posterior wall diastolic velocity and the mitral diastolic closure rate

On the data from 29 subjects (Appendix), posterior wall diastolic velocity/...

velocity was found to correlate positively and significantly with the mitral diastolic closure rate ($r = 0.53$; $P < 0.005$): when patients with mitral stenosis were excluded, the correlation was no longer significant ($r = 0.19$; $P > 0.10$).

Features of the posterior wall echogram

Except in mitral stenosis where the transition to the phase of slow outwards displacement was more gradual (Fig. 8,5), the initial phase of rapid posterior wall displacement terminated abruptly (Figs. 8,1 and 8,8). In rheumatic mitral incompetence with atrial fibrillation, posterior wall diastolic velocity did not very with the length of diastole (Fig. 8,6).

Discussion

It is established that in animals there is an early diastolic phase of rapid left ventricular filling and a subsequent phase of reduced inflow (Nolan et al., 1969; Rushmer, 1972). Angiographic observations (Dodge and Baxley, 1969) and the diastolic waveform of both the apex cardiogram (Kumar and Spodick, 1970) and the mitral echogram (Fig. 2,5) indicate that the normal human left ventricle has similar filling characteristics.

In diastole, the normal left ventricular posterior wall echogram is initially rapidly and subsequently slowly displaced posteriorly (Fig. 8,1), suggesting that posterior wall motion reflects ventricular filling. However, this study has shown that the relation between left ventricular filling and the initial diastolic velocity of the posterior wall is not a simple one. Thus, although posterior wall diastolic velocity was positively and significantly correlated with stroke volume and was reduced when left ventricular inflow was obstructed, it was usually normal in lesions in which the volume and rate of left ventricular filling are characteristically increased, and in individual cases was normal despite a high stroke volume. Similarly, Gibson and Brown (1973) found that the rate of increase of the ultrasonic dimension, which they have used as a measure of left ventricular filling/...

filling, is normal in aortic regurgitation. These discrepancies can be explained; for a given diastolic inflow, the increase in ventricular dimensions and the displacement of the posterior wall will be determined in part by the size and diastolic compliance of the left ventricle.

It has been suggested that early left ventricular filling occurs under the influence of myocardial diastolic recoil (Rushmer, 1972). It is possible that the velocity measured in this study is the velocity of diastolic recoil and that the elastic properties of the myocardium are responsible for the abrupt deceleration in mid-diastole. However, unless myocardial elasticity is impaired in rheumatic heart disease, the pattern of posterior wall motion in mitral stenosis is against this interpretation as is the failure of posterior wall diastolic velocity to correlate significantly with ejection fraction.

Fogelman et al. (1972) measured posterior wall diastolic velocity in normal subjects: the results ranged from 70 to 130 mm/sec. and are similar to those in the present normal group. They postulated that the relatively low velocities which they observed in exercising ischaemic subjects might reflect reduced ventricular compliance. While it is theoretically possible that reduced ventricular compliance could be inferred when a normal or high stroke volume and a reduced posterior wall diastolic velocity are associated, this study has shown that posterior wall diastolic velocity is unlikely to be a useful index of compliance in the dilated ventricle.

On the present evidence it must be concluded that the initial diastolic velocity of the left ventricular posterior wall has no practical value as an index of left ventricular filling.

Ultrasonic assessment of left ventricular function in acute myocardial infarction

The dimensions, volumes and ejection fraction of the left ventricle were measured/...

measured by ultrasound in 10 patients with acute myocardial infarction. The infarct was anterior in 4 and inferior in 6 instances. 4 patients had clinical and radiographic evidence of moderate left heart failure (Table 8,3): of these, one (Case 2) had long-standing cardiomegaly from chronic renal failure and systemic hypertension, one (Case 4) had clinical signs of acute papillary muscle dysfunction and in a third (Case 1), the clinical diagnosis of interventricular septal rupture was confirmed at post-mortem. The ultrasound examinations were carried out on the second or third day after infarction.

The results, which were compared with those from 13 normal subjects (Tables 8,1 and 8,2), have been tabulated - Table 8,3.

Although the reductions in ejection fraction ($P<0.02$) and posterior wall diastolic velocity ($P<0.01$) were the only significant abnormalities in the group as a whole, other abnormalities were observed in irdividual patients.

This was a small, preliminary study and no general conclusions on ventricular performance in myocardial infarction with left heart failure can be drawn from it. Thus, the patients in failure were atypical in that one had pre-existing cardiac enlargement and three had a cause for left ventricular volume overload. Only Case 3, who had no history of previous cardiac disease and in whom there was no evidence of mitral regurgitation or intra-cardiac shunting, conformed to the more usual clinical picture.

An increase in left ventricular size is common in acute myocardial infarction (Karliner and Ross, 1971): in the present series, the ultrasonic dimension and the derived end-diastolic volume were marginally elevated in two patients with uncomplicated infarction and were grossly increased in the patients with papillary muscle dysfunction (Case 4) and established cardiomegaly (Case 2). The findings suggest that an increase in left ventricular size is not an invariable accompaniment of myocardial infarction or of complicating left heart failure and that left ventricular enlargement may be present without evidence/...

evidence of heart failure.

Scheidt et al. (1973) found that while stroke volume and cardiac output may be reduced in infarction complicated by mild or moderate failure, they are normal in uncomplicated infarction. An increased cardiac output has occasionally been observed in acute myocardial infarction (Shillingford and Thomas, 1967). The normal stroke volume and cardiac output in uncomplicated infarction and the reduced values in a single patient with left heart failure (Case 3) are in keeping with the findings of other workers. While the normal values in Case 1 and the high values in Cases 2 and 4 are at variance with the observations of others, they were to be expected in view of the associated cardiac abnormalities. It should, however, be noted that in the presence of mitral regurgitation, stroke volume derived by ultrasound may be very different from true forward stroke volume.

There are no published reports on the ejection fraction of the acutely infarcted ventricle. By the criteria of Kennedy et al. (1966), ejection fraction was abnormal in only two patients (Cases 3 and 4), both of whom were in failure. However, values below the normal range reported in Table 8,2, were observed in several uncomplicated infarcts. Because the ejection fraction of the normal left ventricle requires further study, only the following limited conclusions are possible: in the context of acute myocardial infarction left heart failure may be associated with a normal or a reduced ejection fraction and in uncomplicated infarction the ejection fraction is not substantially reduced.

Posterior wall diastolic velocity was low in a patient with and in others without left heart failure. The low velocities were recorded despite normal or high stroke volumes and could theoretically reflect reduced ventricular compliance.

Although Loeb et al. (1973) have found haemodynamic measurements to be of little value in estimating ventricular performance following myocardial infarction, echocardiographic measurements may have more to offer.

The amplitude of the mitral echogram

The amplitude of the mitral echogram is conventionally measured as the vertical distance between points C and E in Fig. 5, 1 (Edler, 1967).

Published values for the amplitude of the normal mitral echogram vary:-

Amplitude mm.	
25 - 40	Winters et al., 1967
24 - 32	Segal, Likoff and Kingsley, 1966
20 - 33, mean 27	Edler, 1967
14 - 33, mean 22	Ross, 1972
19 - 27, mean 22	Wharton and Lopez Bescos, 1970
23 - 32, mean 28	Burgess et al., 1973

In the study reported in Chap. 2, the echograms selected for measurement of the mitral diastolic closure rate were of maximal amplitude. Echogram amplitudes were measured in the 45 subjects whose diastolic closure rates have been graphed in Fig. 2, 7: the range was 22 to 41 mm with a mean of 29.1 $\pm$ 4.3 mm. These are higher values than most of those previously reported and they may indicate that other workers have been less careful to record the maximal echogram amplitude.

Chakorn et al., 1972 pointed out that the mitral echogram is a composite of mitral ring movement and of movement of the anterior cusp in relation to the ring: Fig. 4,6 illustrates this well. Conventionally, the total amplitude of the mitral echogram, which includes the contribution of ring movement in systole, is measured. It may, however, on occasion be interesting to measure diastolic excursion of the anterior leaflet i.e. the vertical distance between points D and E in Fig. 5,1. In the normal subjects (Fig. 2, 7) this value ranged from 20 to 36.5 mm with a mean of 24.0 $\pm$ 3.8 mm.

Wharton and Lopez Bescos (1970) found an increased mitral echogram amplitude in aortic and mitral regurgitation and postulated that left ventricular dilatation might be responsible. Bellhouse (1972) used these findings to support/...

support his own observations on a model mitral valve: he suggested that in a dilated ventricle, reduced vortex strength favours increased diastolic excursion of the mitral leaflets. Chakorn et al. (1972) concluded that increased left ventricular stroke volume is associated with increased echogram amplitude and that increased ring movement is the most important factor contributing to the increased overall excursion of the echogram. On the grounds that echogram amplitude is little different from normal in mitral stenosis (Fig. 5,4) and that normal or low amplitudes have been observed in ischaemic mitral incompetence (Table 7,2) and in aortic regurgitation (Fig. 6,1), these conclusions were thought to be suspect.

The relations of echogram amplitude to the stroke volume, end-diastolic volume, end-systolic volume and ejection fraction of the left ventricle were investigated in 46 subjects. Of these 13 were normal, 8 had aortic regurgitation, 10 had non-rheumatic mitral regurgitation and 15 were on regular dialysis therapy (Appendix): patients with rheumatic mitral valve disease were excluded from the analysis. Amplitude and stroke volume were not significantly correlated ($r = -0.50$; $P>0.10$ in non-rheumatic mitral regurgitation and $r = -0.25$; $P>0.10$ in the remaining 36 patients). Amplitude and both end-diastolic volume ($r = -0.40$; $P<0.01$ - Fig. 8,9) and end-systolic volume ($r = -0.39$; $P<0.02$) were negatively and significantly correlated. The correlation between amplitude and ejection fraction ($r = 0.29$; $P<0.05$) was positive and significant. The findings indicate that as the left ventricle dilates, mitral echogram amplitude decreases: as suggested in Chap. 7, chordal stretching in diastole with consequent restriction of mitral leaflet excursion may be responsible.

Summary

The literature on the ultrasonic dimension of the left ventricle and on the ventricular volumes and ejection fraction derived from it is reviewed.

The results of an echocardiographic study of the dimensions, volumes and ejection fraction of the left ventricle in normal subjects and in patients with aortic and mitral valve disease are reported. It is concluded that even in the presence/...

presence of ventricular dilatation, the cube of the ultrasonic dimension provides a useful estimate of left ventricular volume. Attention is drawn to three possible sources of measurement error and to the need for further echocardiographic studies of the ejection fraction.

The relation between left ventricular filling and the initial diastolic velocity of the left ventricular posterior wall was shown not to be a simple one. It is concluded that measurement of the posterior wall diastolic velocity is of little practical value in the assessment of left ventricular filling.

In a series of normal subjects mitral echogram amplitude was 29.1 ± 4.3 mm. No correlation was observed between echogram amplitude and the mitral diastolic closure rate. Amplitude and the end-diastolic and end-systolic volumes of the left ventricle were negatively and significantly correlated. The correlation between amplitude and ejection fraction was positive and significant.

TABLE 8,1

LEFT VENTRICULAR DIMENSIONS AND VOLUMES

		Normal	Mitral Stenosis (pre-operative)	Mitral Stenosis (post-operative)	Mitral Stenosis + regurgitation	Mitral regurgitation (ischaemic)	Mitral regurgitation (non-ischaemic)	Aortic stenosis	Aortic regurgitation
End-diastolic dimension cm.	n	13	8	8	4	7	3	3	12
	Range	3·8 - 5·3	3·8 - 5·5	3·8 - 5·5	4·9 - 6·7	5·2 - 7·8	5·5 - 5·7	4·4 - 5·4	5·0 - 7·5
	Mean	4·7	4·6	4·9	5·6	6·4	5·6	4·8	6·4
	SD	0·50	0·61	0·63	0·85	0·91	0·10	0·51	0·71
	P		> 0·10	> 0·10	< 0·10	< 0·001	< 0·02	> 0·10	< 0·001
End-diastolic volume ml.	n	13	8	8	4	7	3	3	12
	Range	53 - 146	56 - 164	53 - 164	119 - 297	143 - 469	166 - 184	85 - 158	125 - 422
	Mean	109	104	120	188	275	176	113	267
	SD	31	41	41	85	119	9	40	85
	P		> 0·10	> 0·10	< 0·10	< 0·005	< 0·005	> 0·10	< 0·001
End-systolic dimension cm.	n	13	8	8	4	7	3	3	12
	Range	1·9 - 3·5	2·4 - 3·9	2·4 - 4·3	2·7 - 5·0	4·5 - 5·9	3·4 - 4·1	2·1 - 3·5	2·9 - 6·3
	Mean	2·8	3·0	3·4	4·0	5·1	3·8	3·0	4·2
	SD	0·51	0·57	0·63	1·2	0 5	0·35	0·77	1·0
	P		> 0·10	< 0·025	< 0·10	< 0·001	< 0·005	> 0·10	< 0·001
End-systolic volume ml.	n	13	8	8	4	7	3	3	12
	Range	6 - 42	13 - 59	14 - 81	19 - 125	89 - 208	41 - 68	9 - 44	25 - 244
	Mean	23	30	43	75	133	57	30	87
	SD	12	17	21	55	41	15	18	62
	P		> 0·10	< 0·025	< 0·10	< 0·001	< 0·005	> 0·10	< 0·005
Heart rate /min.	n	11	7	8	5	7	3	3	12
	Range	55 - 100	50 - 75	65 - 100	54 - 89	56 - 100	77 - 120	63 - 86	58 - 88
	Mean	74	65	77	74	82	89	74	69
	SD	15	9	12	13	16	12	3	10
	P		> 0·10	> 0·10	> 0·10	> 0·10	< 0·10	> 0·10	> 0·10

TABLE 8,2

LEFT VENTRICULAR FUNCTION

		Normal	Mitral Stenosis (pre-operative)	Mitral Stenosis (post-operative)	Mitral Stenosis + regurgitation	Mitral regurgitation (ischaemic)	Mitral regurgitation (non-ischaemic)	Aortic stenosis	Aortic regurgitation
Stroke volume ml.	n	13	8	8	4	7	3	3	12
	Range	46 - 121	42 - 117	39 - 112	85 - 172	54 - 262	98 - 138	59 - 114	100 - 291
	Mean	86	74	76	114	142	119	83	181
	SD	23	28	24	39	86	20	29	53
	P		>0·10	>0·10	>0·10	>0·10	<0·05	>0·10	<0·001
Cardiac output l/min.	n	11	7	8	4	7	3	3	12
	Range	3·1 - 9·6	2·9 - 8·8	3·3 - 8·0	4·9 - 12·5	4·9 - 26·0	9·8 - 10·9	3·7 - 8·3	7·7 - 19·5
	Mean	6·5	5·0	5·8	7·6	11·3	10·4	6·2	12·5
	SD	2·2	2·0	1·8	3·5	7·2	0·6	2·3	3·8
	P		>0·10	>0·10	>0·10	>0·10	<0·02	>0·10	<0·001
Ejection fraction	n	13	8	8	4	7	4	3	12
	Range	0·70 - 0·89	0·62 - 0·87	0·50 - 0·74	0·44 - 0·85	0·32 - 0·65	0·59 - 0·77	0·61 - 0·90	0·37 - 0·84
	Mean	0·80	0·72	0·65	0·65	0·49	0·67	0·74	0·70
	SD	0·065	0·09	0·08	0·18	0·12	0·09	0·14	0·14
	P		<0·10	<0·001	>0·10	<0·001	<0·05	>0·10	<0·05
Posterior wall excursion mm.	n	20	10	8	5	8	3	3	11
	Range	10·0 - 15·0	7·5 - 13·5	7·5 - 12·0	13·0 - 8·0	6·5 - 11·5	10·0 - 12·5	9·5 - 13·0	9·5 - 15·5
	Mean	12·1	10·4	9·9	14·7	8·2	11·7	10·8	12·7
	SD	1·3	2·1	1·4	2·7	2·0	1·4	1·9	2·0
	P		<0·025	<0·001	<0·10	<0·001	>0·10	>0·10	>0·10
Posterior wall diastolic velocity mm/sec.	n	17	10	7	5	2	3	3	10
	Range	76 - 156	24 - 54	24 - 133	33 - 107	128 - 154	62 - 216	90 - 190	90 - 190
	Mean	114	40	53	73	141	143	77	121
	SD	28	9	38	28	13	77	38	38
	P		<0·001	<0·001	<0·01	<0·10	>0·10	>0·10	>0·10

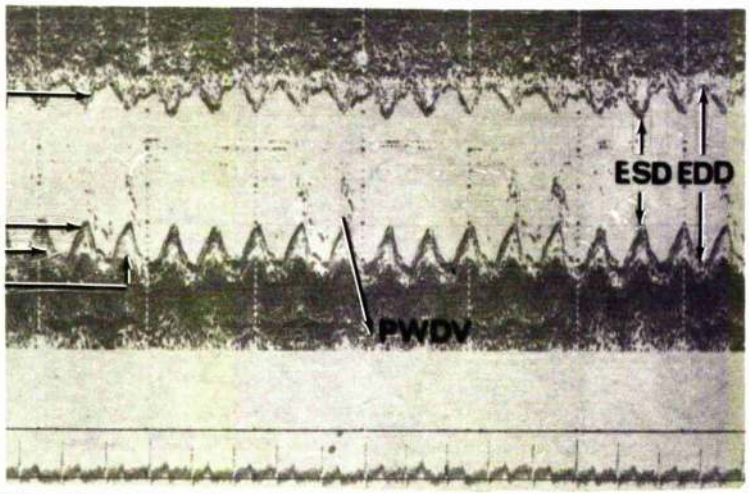


FIG. 8,1 LEFT VENTRICULAR DIMENSION AND POSTERIOR WALL DIASTOLIC VELOCITY - METHODS OF MEASUREMENT

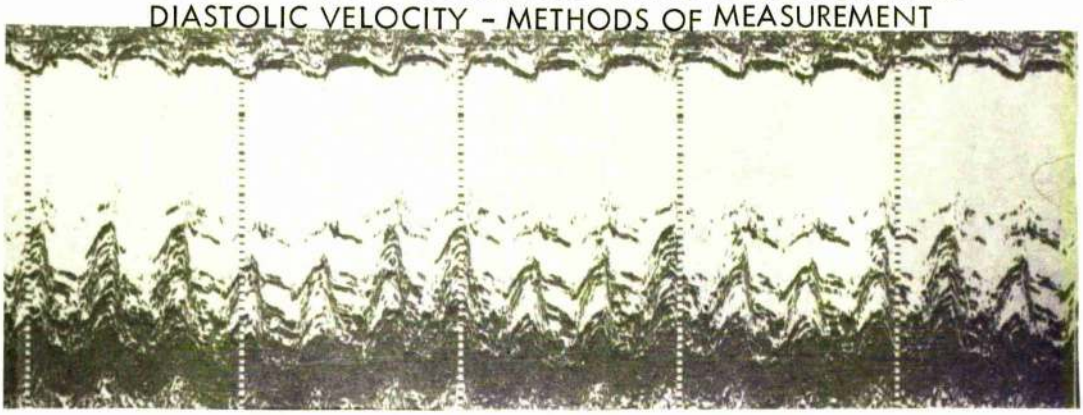


FIG. 8,2 VARYING POSTERIOR WALL EXCURSION

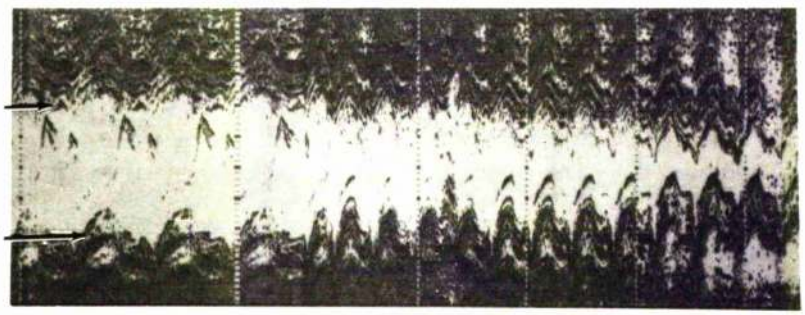


FIG. 8,3 SWEEP FROM MITRAL VALVE TO APEX OF LEFT VENTRICLE

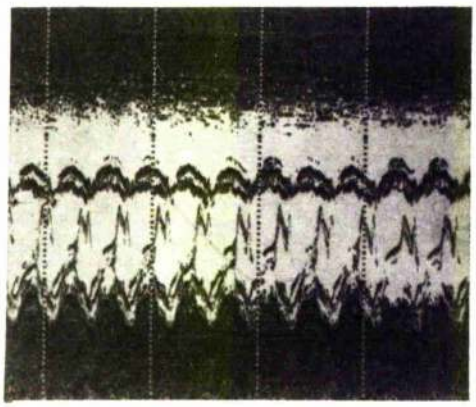


FIG. 8,4 REVERSED SEPTAL MOTION IN ATRIAL SEPTAL DEFECT
ESV = 12 ml EF = 0.84

	Infarct site	LVF	Heart rate	EDD cm.	EDV ml.	ESD cm.	ESV ml.	SV ml.	CO l/min	EF	PWE mm.	PWDV mm/sec.
1	Anterior	+	95	4·7	102	2·4	15	87	8·3	0·85	13·0	106
2	Anterior	+	65	6·3	244	3·8	53	191	13·0	0·78	13·0	105
3	Inferior	+	80	4·9	115	4·0	64	51	4·1	0·44	10·0	
4	Inferior	+	88	6·9	334	5·7	180	154	13·6	0·46	9·5	47
5	Anterior	-	75	5·0	125	2·9	24	101	7·6	0·80	16·0	112
6	Anterior	-	92	5·0	125	3·4	39	86	7·9	0·69	16·5	100
7	Inferior	-	100	5·0	125	3·5	42	83	8·3	0·66	16·0	53
8	Inferior	-	86	5·3	151	3·3	34	117	10·1	0·77	15·0	60
9	Inferior	-	80	3·6	48	2·7	20	28	2·2	0·58	10·5	73
10	Inferior	-	75	5·4	157	3·8	54	103	7·7	0·66	10·5	45
	Mean		84	5·2	153	3·6	53	100	8·3	0·67	13	78
	SD		11	0·89	81	0·91	47	47	3·5	0·14	2·75	28
	P				>0·10		<0·10	>0·10		<0·02		<0·01

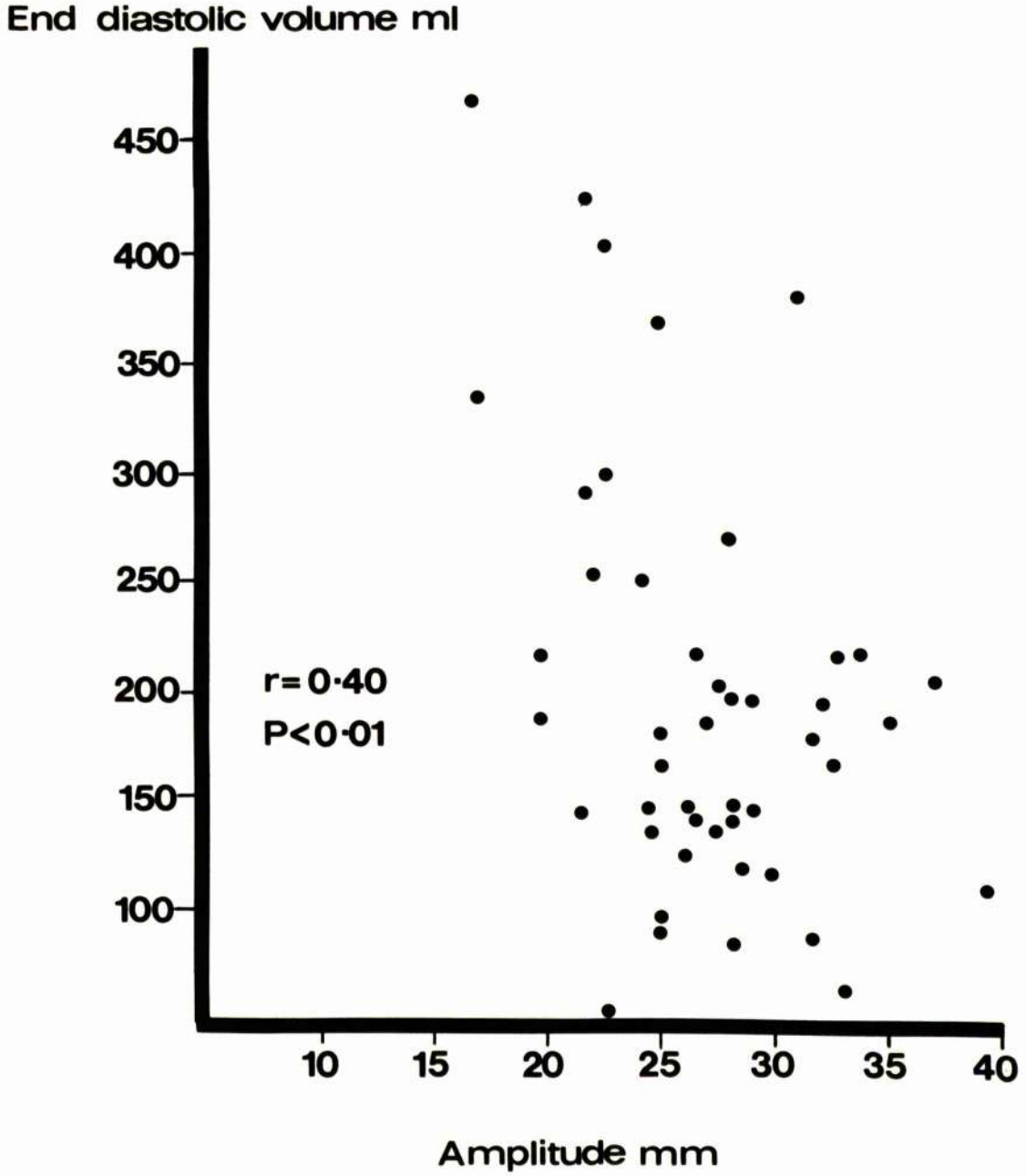


FIG. 8,9 LEFT VENTRICULAR END-DIASTOLIC VOLUME AND MITRAL ECHOGRAM AMPLITUDE - 46 PATIENTS

CHAPTER 9

ULTRASONIC ASSESSMENT OF LEFT VENTRICULAR FUNCTION IN CHRONIC RENAL FAILURE

As chronic renal failure progresses to the point where regular dialysis therapy (RDT) is necessary, fluid overload, systemic hypertension (Curtis et al., 1969) and anaemia (Adamson, Eschbach and Finch, 1968) are usual. Clinical features of circulatory congestion are common in acute (Agrest and Finkielman, 1967) and chronic (Del Greco, Simon and Roguska, 1969) renal failure. Although Del Greco et al. (1969) have suggested that the circulatory congestion has a non-cardiac basis, others have postulated that myocardial toxins circulate in uraemic patients and that impaired myocardial contractility may therefore be a contributory factor (Bailey, Hampers and Merrill, 1967).

Limited haemodynamic studies have been carried out in acute renal failure (Agrest and Finkielman, 1967) and in patients on maintenance haemodialysis: most have demonstrated an increased cardiac output (Goss et al., 1967; Del Greco et al., 1969; Hampers et al., 1969; Gutkin et al., 1969; Neff et al., 1971; Tuckman et al., 1972). Systolic time intervals have been measured in dialysis patients without circulatory congestion (Prakash and Wegner, 1972) but there are no published reports on left ventricular function in patients with circulatory congestion.

Pericardial effusion can be diagnosed (Feigenbaum, 1970) and left ventricular volumes and ejection fraction can be measured by echocardiography (Pombo, Troy and Russell, 1971; Ludbrook et al., 1973).

The aims of the present study were to use the method to investigate left ventricular performance in patients with chronic renal failure, with and without circulatory congestion; to compare the results before and after a short period on RDT with those from normal subjects and patients stabilised on RDT; and to note the incidence of pericardial effusion in dialysis patients.

Methods/...

Methods

The equipment used was an Eskoline 20 ultrasonoscope with an unfocused transducer. Records were made with an ultraviolet strip-chart recorder (Honeywell Visicorder) except in 4 instances where the time-position display was recorded directly from the oscilloscope face by Polaroid photography. The patients were examined supine and the transducer was positioned and angled to display simultaneous echoes from the interventricular septum and from the left ventricular posterior wall below the free margin of the posterior cusp; care was taken to avoid the region of the posterior papillary muscle (Chaps. 4 and 8). Dimensions, volumes and the ejection fraction were measured and calculated from the mean of up to 10 cardiac cycles by the methods described by Pombo et al. (1971). The results were compared with those from 13 normal subjects (Chap. 8). Statistical analysis was by Student's 't' tests for the significance of differences between means and between paired observations.

Patients

Three groups were studied.

Group 1 (Table 9,1) - pre dialysis

10 patients being considered for inclusion in a regular dialysis programme. The clinical features of circulatory congestion were present in each; all had peripheral oedema, raised jugular venous pressure, apical triple rhythm and basal crepitations. Of the 8 patients in whom information was available, 7 had radiographic pulmonary oedema. One patient was normotensive, the remainder had mild or moderate systemic hypertension. All were anaemic, the highest packed cell volume being 31%. The mean values for packed cell volume, blood pressure and cardio-thoracic ratio in this group have been tabulated (Table 9,3).

Patient 9 was rejected for RDT and patient 6 died before it could begin.

Group 2 (Table 9,2) - on RDT

6 patients from Group 1 on RDT for less than 8 (mean 5) weeks. They were dialysed three times per week and ultrasonic examination was carried out on the first or second post-dialysis day. Signs of circulatory congestion were present/...

present in two patients of whom one had radiographic pulmonary oedema. One was normotensive and one was severely hypertersive. All were anaemic, the highest packed cell volume being 23%. The mean values for packed cell volume, blood pressure and cardio-thoracic ratio in this group have been tabulated (Table 9,3).

Group 3 (Table 9,4) - on RDT

A miscellaneous group of 9 patients, all on RDT for more than 6 months but with widely varying clinical features. Thus, patients 3, 4, 11 and 12 were examined two weeks after successful renal transplant procedures; patients 1 and 13 were examined to confirm radiographic evidence of pericardial effusion and patients 14, 15 and 16 were examined because of persisting evidence of circulatory congestion despite RDT.

Signs of circulatory congestion were present in 6 patients of whom 5 had radiographic pulmonary oedema. Two were normotensive and systemic hypertension varied from mild to severe in the remainder. All were anaemic, the highest PCV being 32%. The cardio-thoracic ratio was clearly increased in 6 cases.

Results

The echocardiographic findings in Groups 1, 2, and 3 are presented in Tables 9,5; 9,6 and 9,7 respectively. (Because volume was calculated from dimension corrected to two decimal places, the volumes tabulated are not always the precise cube of the corresponding dimensions).

Group 1 (Table 9,5)

Pericardial effusion was diagnosed from the plain chest radiograph in 3 cases (Table 9,1) and by ultrasound (Fig 9,1) in 6 cases (patients 1 - 4, 6 and 8).

The mean heart rate was 95 beats/min. Mean end-diastolic and end-systolic dimensions and the corresponding mean end-diastolic ($P < 0.001$) and end-systolic ($P < 0.02$) volumes were significantly increased with respect to normal. End-diastolic volume was greater than normal in 8 cases: it was normal in the only patient (patient 3) known not to have pulmonary oedema and was within the normal range/...

range but 1SD above the normal mean in one other (patient 9). End-systolic volume was normal in 4 cases, including patients 3 and 9; was within the normal range but 1SD above the normal mean in one and was abnormally increased in the remainder.

Mean stroke volume and mean cardiac output were increased ($P < 0.001$): values within the range of the present normal series were, however, recorded in patients 3 and 9 for stroke volume and in patient 9 for cardiac output. Although the mean value was normal, ejection fraction was reduced in two patients (5 and 7).

Cardiac output and packed cell volume were not inversely correlated ($r = 0.24$; $P > 0.10$).

Group 2 (Table 9,6)

Pericardial effusion was diagnosed from the plain chest radiograph in one case (Table 9,2) and by ultrasound in two cases (patients 4 and 8).

The mean heart rate was 80 beats/min. Mean end-diastolic and end-systolic dimensions and the corresponding mean end-diastolic ($P < 0.005$) and end-systolic ($P < 0.001$) volumes were increased with respect to normal: the volumes were greater than normal in all but patient 3.

Mean stroke volume and mean cardiac output were above normal ($P < 0.02$): stroke volume was, however, normal in patient 3 and cardiac output was normal in patients 3 and 10. Two patients (3 and 7) had ejection fractions below the present normal range and mean ejection fraction for the group was reduced ($P < 0.01$). The findings in patients with and without circulatory congestion are compared in Fig. 9,2.

Cardiac output and packed cell volume were not inversely correlated ($r = 0.21$; $P > 0.10$).

Comparison of findings before RDT and after 5 weeks on RDT (Groups 1 and 2)

With RDT, the heart rate ($P < 0.02$) and the cardiothoracic ratio ($P < 0.05$) decreased (Table 9,3). Alterations in left ventricular volumes and performance were/...

were not significant and showed no consistent trends (Table 9,6).

In two patients (7 and 8) signs of circulatory congestion persisted at the second examination despite a period of RDT: cardiac output and end-diastolic volume increased in both.

In four patients (3,4,5 and 10) signs of circulatory congestion resolved between the examinations. While cardiac output fell in three instances with an accompanying reduction in end-diastolic volume in patients 3 and 10, it rose in patient 5 in whom end-diastolic volume was constant.

Echocardiograms and chest radiographs from patients 5 and 10 before and after RDT are shown in Figs. 9,3 and 9,4.

Group 3, (Table 9,7)

Pericardial effusion was diagnosed from the plain chart radiograph in 4 cases (Table 9,4) and by ultrasound in patients 1, 11 and 12.

Because the results varied widely, mean values were not calculated for this group. In Fig. 9,2 the results are compared with those from the normal series and from Groups 1 and 2: patients with clinical signs of circulatory congestion are identified.

End-diastolic and end-systolic volumes were increased in five and normal in four cases; cardiac output was high in five, normal in three and low in one case; stroke volume was high in three, normal in five and low in two cases; ejection fraction was normal in five and reduced in four cases.

Of the six patients in this group with signs of circulatory congestion, four had a reduced ejection fraction and three had a low cardiac output. Packed cell volume and cardiac output were not inversely correlated ($r = 0.53$; $P > 0.10$).

The echocardiogram and chest radiograph of patient 15 are shown in Fig. 9,5.

Discussion

Cardiac output has been found to be high in acute renal failure (Agrest and/...

and Finkielman, 1967) and in patients stabilised on maintenance haemodialysis (Goss et al., 1967; Gutkin et al., 1969; Neff et al., 1971; Tuckman et al., 1972). The present study has shown that a high output is also usual in patients with chronic renal failure awaiting their first dialysis or on RDT for a short period. It confirms the findings in a group of patients about to start RDT in whom cardiac output was measured by dye-dilution (Table 9,8). It should, however, be noted that cardiac output was lower in the group in which it was measured by conventional methods.

A high cardiac output is theoretically to be expected in end-stage chronic renal failure which is frequently associated with anaemia and an increased plasma volume (Goss et al., 1967; Gutkin et al., 1969). Neff et al. (1971) concluded that the increased cardiac output in chronic renal failure is secondary to anaemia while Gutkin et al. (1969) and Tuckman et al. (1972) demonstrated a significant inverse correlation between packed cell volume and cardiac output in patients stabilised on RDT. The absence of such a correlation in the present series of patients, all of whom were anaemic, suggests that before and early in the course of RDT, the high cardiac output has additional determinants. One of these may be increased blood volume; total body water is increased in patients about to start on RDT (Table 9,8), plasma volume is increased in acute renal failure and in patients on maintenance haemodialysis (Gutkin et al., 1969), while blood volume and cardiac output are significantly correlated in acute renal failure (Agrest and Finkielman, 1967). The presence of a surgically created arterio-venous communication which may increase the cardiac output by as much as 25% (Menno et al., 1967) is another possible but probably less important factor.

Del Greco et al. (1969) observed widely varying cardiac outputs and both normal and prolonged circulation times in patients with circulatory congestion. They concluded that in chronic uraemia, circulatory congestion results from a variety of haemodynamic, myocardial and metabolic alterations. Left ventricular function has been assessed in more detail in the present study.

The/...

The end-diastolic volume and the contractility of a ventricle determine its ejection fraction. The ejection fraction thus provides information on ventricular performance but is not an adequate index of contractivity (Mitchell and Wildenthal, 1972). Ultrasonic and cineangiographic estimates of ejection fraction are closely correlated (Pombo, Troy and Russell, 1971). Although in the present normal series the ejection fraction was never less than 70%, others have accepted 52% as the lower limit of normal (Kennedy et al., 1966; Gibson and Brown, 1973).

Before RDT, circulatory congestion was associated with high stroke and end-diastolic volumes and with a normal ejection fraction. There were, however, two exceptions; in patients 5 and 7 stroke volume was increased but the ejection fractions were well below the present normal range. Although they fall within the normal limits of Kennedy et al. (1966), it is suggested that these values are inappropriately low for ventricles functioning at a high end-diastolic vomume and that they therefore reflect impaired left ventricular function. It is impossible to be certain whether impaired contractility or an increase in end-diastolic volume to the point where the ventricles were operating on the downslope of the Frank-Starling curve was responsible for the reduced ejection fractions. However, the fact that after RDT, ejection fraction increased despite unaltered (patient 5) and increased (patient 7) end-diastolic volumes, suggests that factors other than the Frank-Starling mechanism were operative.

The findings in patients in whom circulatory congestion was present despite more than 6 months on RDT were clearly different. Thus though cardiac output was high and ventricular function was normal in 3 patients, in three others stroke volume was low and ejection fraction was severely reduced. End-diastolic volume was increased in patient 15 (Fig. 9,5) who conformed clinically to Bailey's description of uraemic cardiomyopathy (Bailey et al., 1967). Although he had no clinical evidence of ischaemic heart disease and was only moderately hypertensive, cardiac enlargement had progressed rapidly and he was in intractable congestive cardiac failure with hepatomegaly and ascites/...

ascites. End-diastolic volume was normal in patient 14 but cardiac size increased subsequently and she died in left heart failure: there was no macroscopic evidence of coronary artery disease at autopsy. Patient 16 had a history of angina and frequent arrhythmias during dialysis procedures.

It can be broadly concluded firstly, that circulatory congestion and pulmonary oedema before or in the early stages of RDT are usually associated with normal left ventricular performance and secondly, that these features commonly reflect impaired ventricular function in patients maintained on dialysis for longer periods. The findings in Groups 1 and 2 also suggest that in the early stages of RDT, resolution of circulatory congestion is associated with a reduction in cardiac output.

Blood pressure usually comes under satisfactory control within the first two months of RDT (Curtis et al., 1969). Decreases in total exchangeable sodium and extracellular fluid volume have been shown to be associated with the fall in blood pressure on dialysis (Blumberg et al., 1967). In the present series, blood pressure responded variably and in three cases actually increased during the first two months on dialysis. These atypical responses could explain the absence of consistent charges in ventricular dimensions and function over the period but there were other possible variables. Thus, in Group 2, the degree of sodium and water retention could be expected to be greater in those patients examined on the second than in those examined on the first post-dialysis day. Further, all patients had a radial arterio-venous fistula of the Cimino-Brescia type but the size of the communication judged from the dimensions of the arterialised vein varied considerably.

It is interesting to note that in patients 3 and 4 no major haemodynamic adjustments apparently occurred after the first few weeks on RDT: stroke volume and cardiac output were essentially the same when they were examined after less than two months and after a year on dialysis. Fig. 9,6 is a tracing from patient 4 at 12 months: it shows the persistent increase in end diastolic dimension.

Echocardiography/...

Echocardiography is now widely accepted as the method of choice for the detection or exclusion of pericardial effusion (Feigenbaum, 1970): in the present study its superiority over the plain chart radiograph was clearly demonstrated. Pericardial effusion was present in all the Groups studied but its incidence was highest (60%) in the pre-dialysis patients. The effusions diminished or resolved during the early weeks of RDT, the process being reflected in a significant fall in cardio-thoracic ratio despite unaltered ventricular dimensions.

The non-invasive nature of echocardiography suits it to the investigation of patients with renal failure. There are many reasons why such patients should not be subjected to more aggressive investigative methods; they cannot afford to lose blood, they are unable to excrete contrast media and they require intact vessels for access to the circulation at dialysis.

This study has shown that cardiac ultrasound can provide information which is potentially useful for the management of patients on regular dialysis therapy. Thus, apart from the obvious value of identifying or excluding pericardial effusion as a cause of radiographic cardiomegaly, it may be possible to differentiate echocardiographically between patients in whom circulatory congestion could be expected to respond to digitalisation and those in whom it should be treated by an increase in dialysis-time.

The basic, somewhat academic question of whether "uraemia" can by itself impair myocardial contractility remains unanswered. A further study is planned in which multiple assessments of left ventricular function by ultrasound and by systolic time intervals will be made during the first few weeks on dialysis: it will include measurements across single dialysis procedures.

Summary

Signs of circulatory congestion are common in chronic renal failure but their cause has not been established. The dimensions, stroke volume and ejection fraction of the left ventricle were measured by ultrasound in 10 patients about to start RDT. In 6 of them, the measurements were repeated after less than 2 months on RDT. The results were compared with those from normal subjects and from patients on RDT for more than 6 months.

The findings before and after 2 months on RDT were essentially the same: end-diastolic, end-systolic and stroke volumes were increased with respect to normal and, despite radiographic evidence of pulmonary venous congestion and pulmonary oedema, ejection fraction was normal. In contrast, ejection fraction was reduced in 4 of the 6 patients in whom signs of circulatory congestion were present despite more than 6 months on RDT.

It is concluded that whereas left ventricular dysfunction is not usually responsible for circulatory congestion before RDT or in its early stages, impaired ventricular performance should be suspected when circulatory congestion is present in a patient established on RDT for some months.

TABLE 9,1

CLINICAL FEATURES - GROUP 1

	Age/ Sex	PCV %	Blood pressure mm Hg	Mean	Chest radiograph CTR %	Pericardial effusion	Pulmonary oedema	Circulatory congestion	Anti-hypertensive therapy	Renal diagnosis	Progression of renal failure
1	37 F	18	190/90	123	60	?	+	+	+	CGN	Slow
2	32 M	13	140/90	107				+	−	CGN	Rapid
3	19 F	15	180/100	127	67	+	−	+	−	CGN	Rapid
4	23 M	17	180/115	137	65	+	+	+	+	CGN	Slow
5	23 F	24	155/110	125	59		+	+	+	CPN	Slow
6	42 F	18	110/70	83		+	+	+	+	CGN	Rapid
7	46 M	31	155/95	115	66	?	+	+	+	CPN	Slow
8	29 M	24	180/110	133	53	−	+	+	+	Poly arteritis	Slow
9	22 F	30	170/120	137				+	+	Systemic lupus	Slow
10	22 F	15	140/95	110	59	−	+	+	+	CGN	Slow

CGN = Chronic glomerulonephritis CPN = Chronic pyelonephritis

TABLE 9,2 CLINICAL FEATURES - GROUP 2

	Week of RDT	PCV %	Blood pressure mm Hg		Mean	Chest radiograph CTR %	Pericardial effusion	Pulmonary oedema	Circulatory congestion	Anti-hypertensive therapy
3	7	21	145	88	107	57	-	-	—	-
4	5	19	150	90	110	50	?	-	-	+
5	5	17	130	70	90	49	-	-	-	+
7	5	17	200	110	140	51	-	-	+	+
8	3	23	200	140	160	59	+	+	+	+
10	5	18	170	90	117	45	-	-	-	+

TABLE 9,3 COMPARISON OF GROUPS 1 & 2

	PCV%	Blood pressure mm Hg Systolic	Diastolic	Mean	CTR %
Before RDT (n = 10)	$21 \pm 6{\cdot}4$	160 ± 25	100 ± 15	120 ± 17	62 ± 5 (n = 7)
On RDT (n = 6)	$19 \pm 2{\cdot}4$	166 ± 30	98 ± 24	121 ± 25	52 ± 5
P	$>0{\cdot}10$	$>0{\cdot}10$	$>0{\cdot}10$	$>0{\cdot}10$	$<0{\cdot}05$

TABLE 9,4

CLINICAL FEATURES - GROUP 3

	Age/ Sex	PCV %	Blood pressure mm Hg	Mean	Chest radiograph CTR %	Pericardial effusion	Pulmonary oedema	Circulatory congestion	Anti-hypertensive therapy	Renal diagnosis	Time on RDT months
1	37 F	24	170/110	130	68	+	+	+	+	CGN	6
3	19 F	32	130/80	97	58	-	-	-	-	CGN	10
4	23 M	15	160/85	110	51	-	-	-	+	CGN	12
11	29 M	21	210/120	150	70	+	+	+	+	CGN	11
12	34 F	23	110/50	70	70	+	-	-	-	CPN	24
13	30 M	20	240/140	173	51	+	-	+	+	AGN	23
14	39 F	19	190/120	143	58	-	+	+	+	Systemic lupus	12
15	34 M	31	140/110	120	65	-	+	+	+	CGN	7
16	58 M	26	150/80	103	53	-	+	+	-	Polycystic disease	10

AGN Rapidly progressive (acute) glomerulonephritis

TABLE 9,5

ECHOCARDIOGRAPHIC FINDINGS - GROUP 1

	Heart rate min.	End-diastolic dimension cm.	End-diastolic volume ml.	End- diastolic dimension cm.	End- diastolic volume ml.	Stroke volume ml.	Cardiac output l/min.	Ejection fraction	Posterior wall excursion mm.
1	123	5·9	204	2·6	17	187	23·0	0·92	14·5
2	96	5·6	178	3·5	41	137	13·1	0·77	11·0
3	105	4·9	118	2·7	20	98	10·3	0·83	14·5
4	92	6·0	216	3·5	43	173	16·1	0·80	15·5
5	120	6·7	297	5·2	138	159	19·0	0·54	12·5
6	92	5·7	186	2·8	22	164	15·2	0·88	17·0
7	86	6·0	216	4·5	91	125	10·8	0·58	13·0
8	80	5·9	204	3·8	54	150	12·0	0·74	13·5
9	80	5·3	145	3·1	31	114	9·1	0·79	12·0
10	80	5·8	193	3·7	51	142	11·3	0·73	11·0
Mean	95	5·8	195	3·5	51	145	14·0	0·76	13·5
SD	16	0·47	47	0·82	36	28	4·4	0·12	2·0
*P		<0·001	<0·001	<0·02	<0·02	<0·001	<0·001	>0·10	<0·10

*P statistical significance of difference from normal group (Chap. 8)

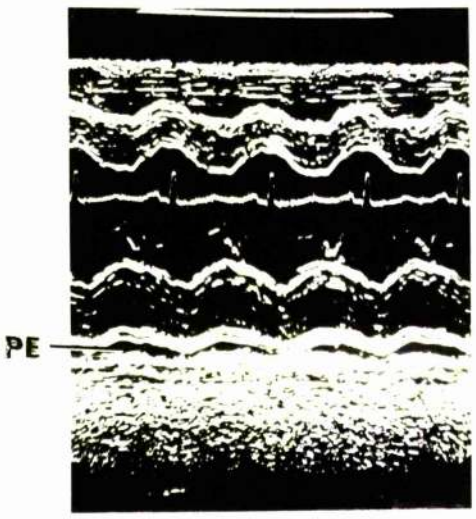
TABLE 9,6 ECHOCARDIOGRAPHIC FINDINGS - GROUP 2

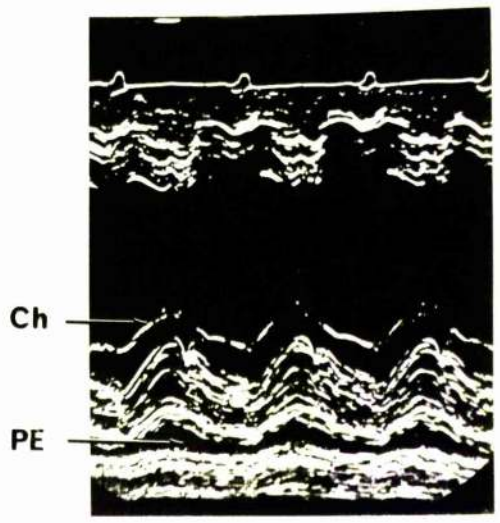
	Heart rate min.	End-diastolic dimension cm.	End-diastolic volume ml.	End-systolic dimension cm.	End-systolic volume ml.	Stroke volume ml.	Cardiac output l/min.	Ejection fraction	Posterior wall excursion mm.
3	86	4·7	103	3·3	34	69	5·9	0·67	10·5
4	67	6·7	302	4·1	69	233	15·6	0·77	15·0
5	104	6·7	297	4·3	76	221	22·9	0·74	16·5
7	86	6·1	224	4·3	82	142	12·2	0·63	18·5
8	75	6·7	297	4·3	82	215	16·1	0·72	18·0
10	64	5·5	169	3·5	42	127	8·2	0·75	12·5
Mean	80	6·1	232	4·0	64	168	13·5	0·71	15·2
SD	15	0·82	82	0·45	21	65	6·1	0·05	3·2
^1P		< 0·005	< 0·005	< 0·001	< 0·001	< 0·02	< 0·02	< 0·01	< 0·05
^2P	< 0·02	> 0·10	> 0·10	> 0·10	> 0·10	> 0·10	> 0·10	> 0·10	> 0·10

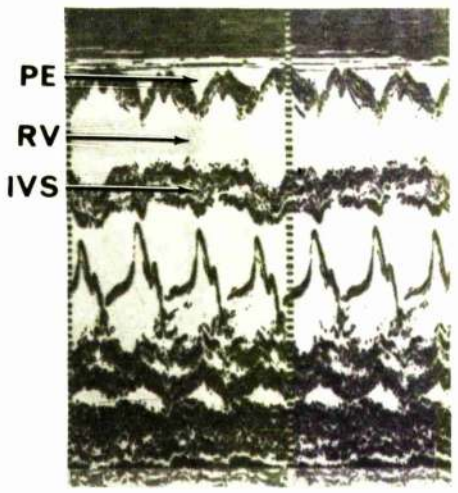
^1P statistical significance of difference from normal group (Chap. 8)
^2P statistical significance of difference from pre-RDT results (Table 8)

TABLE 9,7 ECHOCARDIOGRAPHIC FINDINGS - GROUP 3

	Heart rate min.	End-diastolic dimension cm.	End-diastolic volume ml.	End-systolic dimension cm.	End-systolic volume ml.	Stroke volume ml.	Cardiac output l/min.	Ejection fraction	Posterior wall excursion mm.
1	115	5·7	180	3·2	32	148	17·0	0·82	15·5
3	80	4·6	94	3·1	28	66	5·3	0·70	10·0
4	71	6·6	282	3·6	48	234	16·6	0·83	15·0
11	93	5·5	166	3·8	59	107	10·0	0·64	10·0
12	80	5·3	149	2·8	22	127	10·2	0·85	11·0
13	92	5·2	141	3·3	36	105	9·7	0·74	9·0
14)	113	4·6	94	3·8	55	39	4·4	0·41	7·0
15	100	7·2	369	6·8	319	50	5·0	0·14	7·5
16	71	4·4	86	3·9	58	28	2·0	0·33	8·0







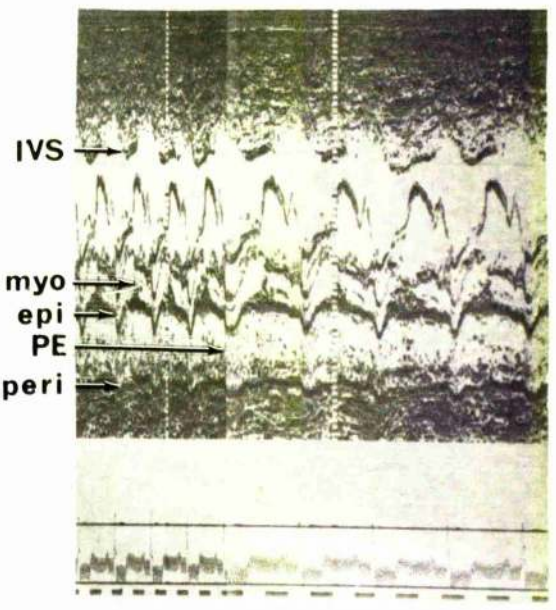


FIG. 9,1 PERICARDIAL EFFUSION IN PATIENTS AWAITING RDT

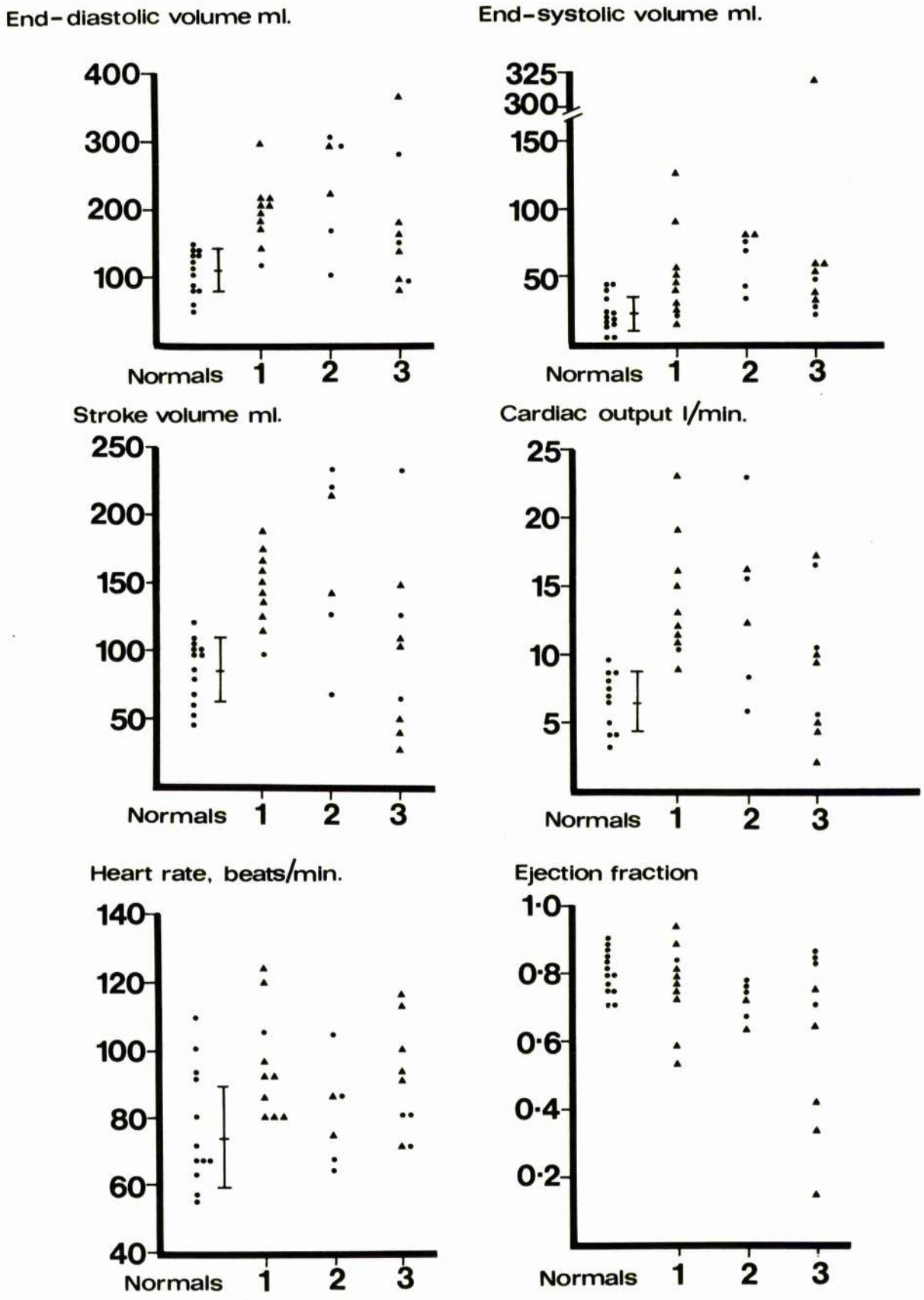
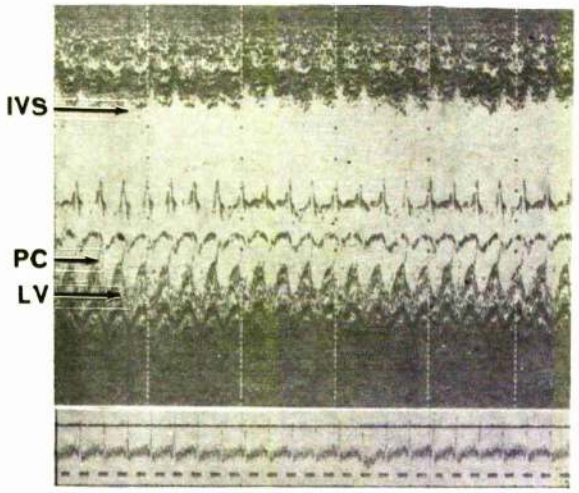
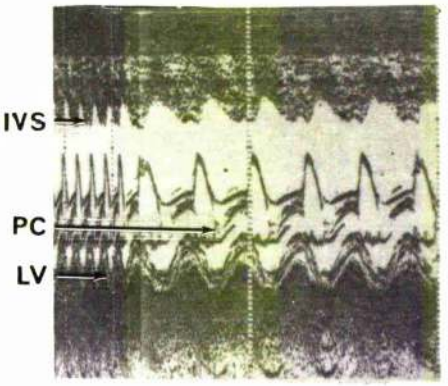


FIG. 9,2 ULTRASONIC ASSESSMENT OF THE LEFT VENTRICLE IN GROUPS 1, 2 and 3 AND IN NORMAL SUBJECTS.

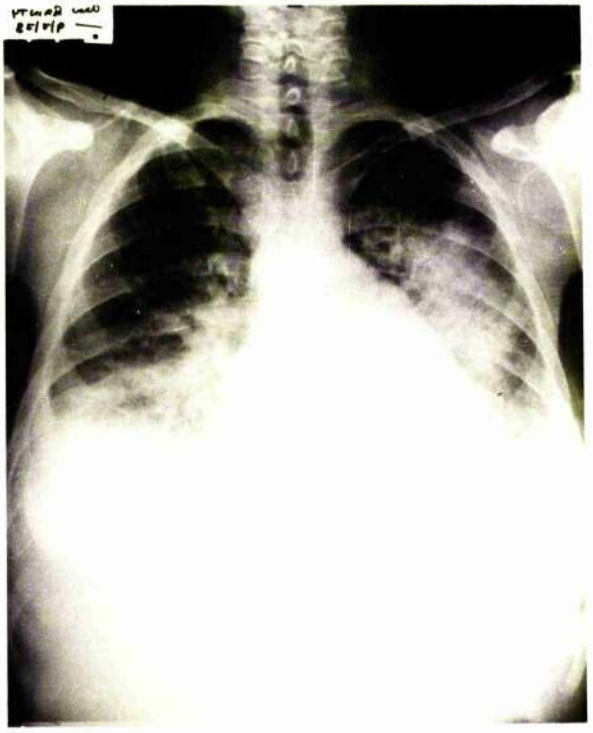
▲ – CIRCULATORY CONGESTION PRESENT



BEFORE RDT



ON RDT



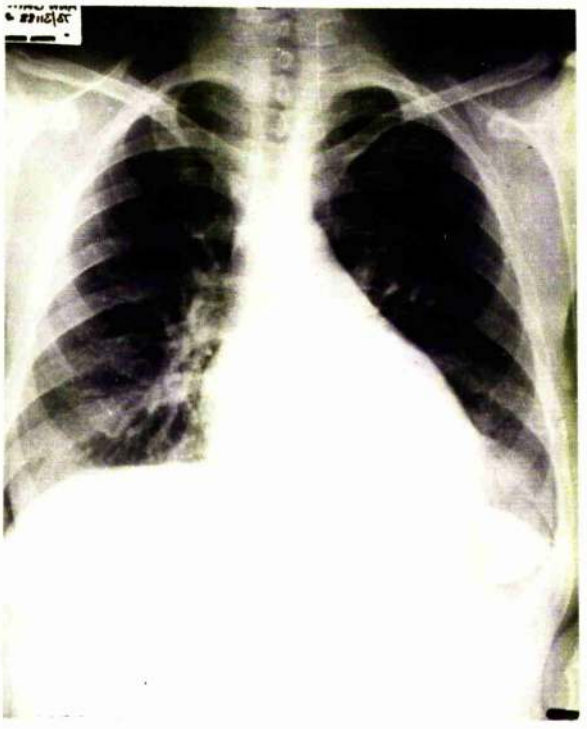
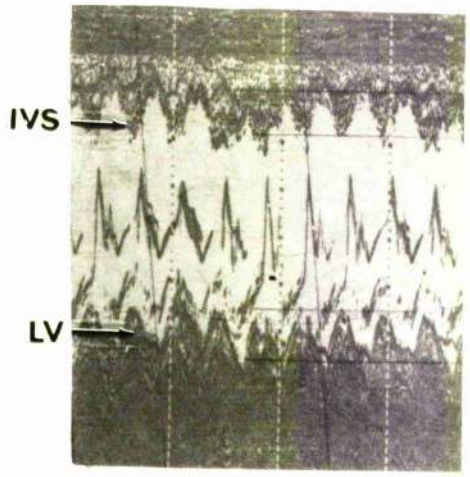
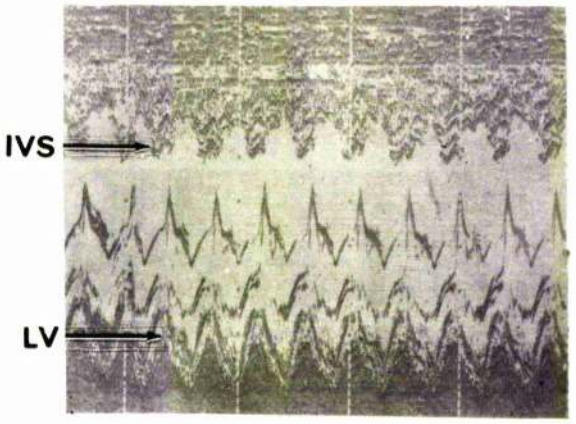


FIG. 9,3 CASE 5



BEFORE RDT



ON RDT

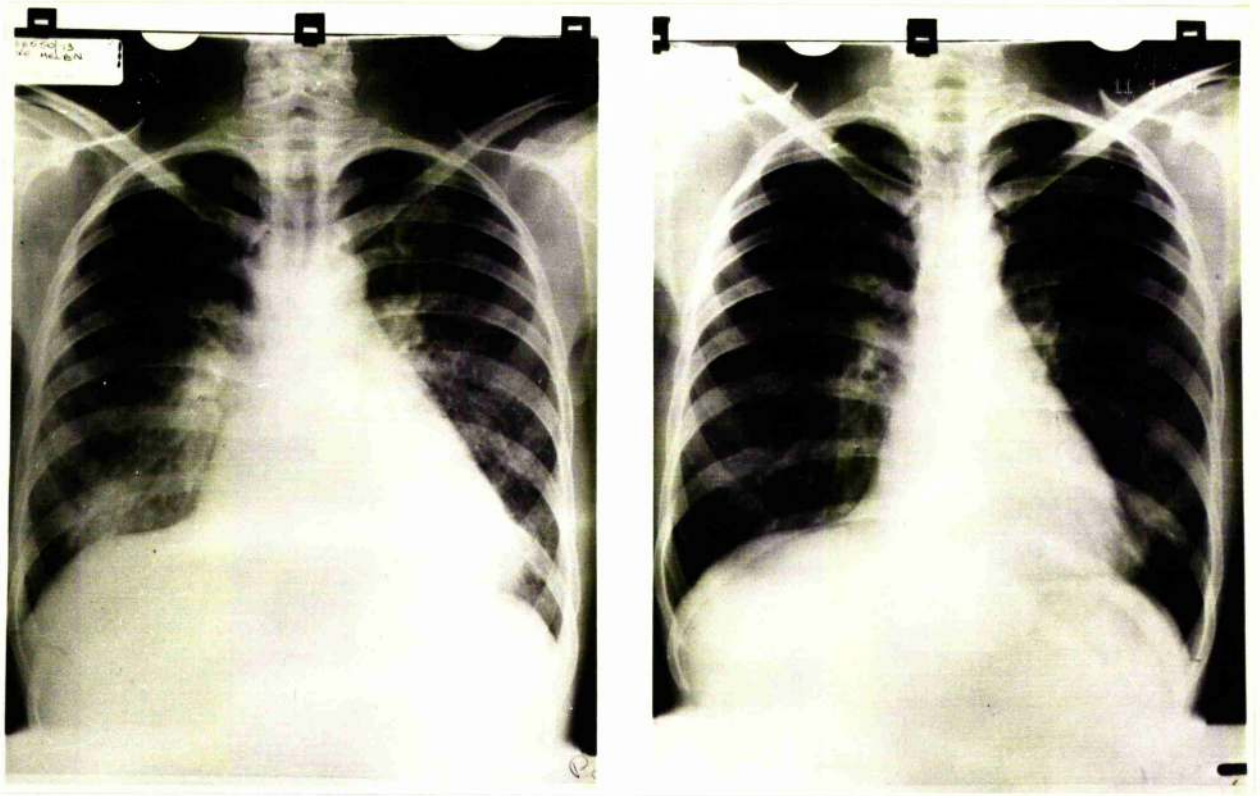
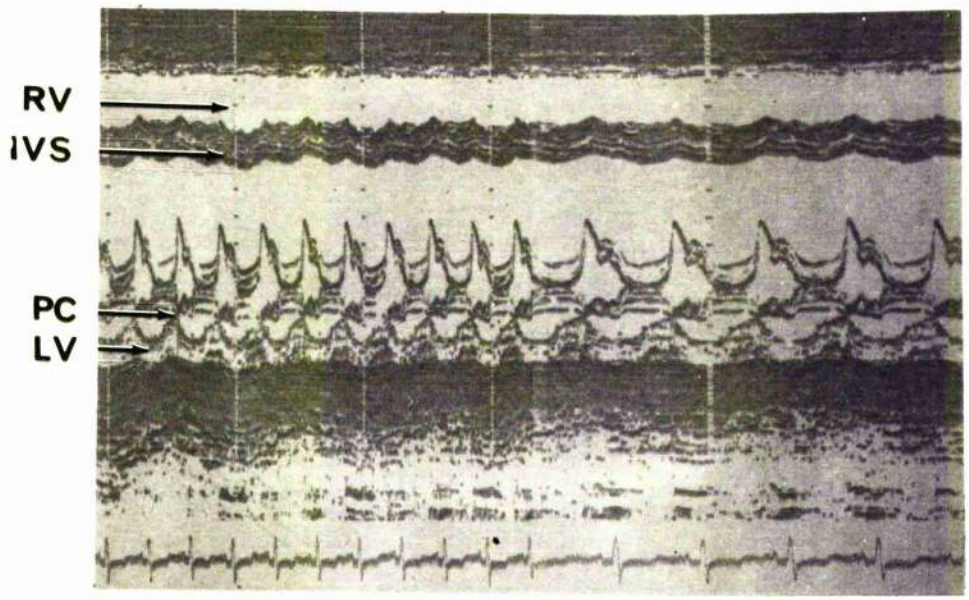


FIG. 9,4 CASE 10



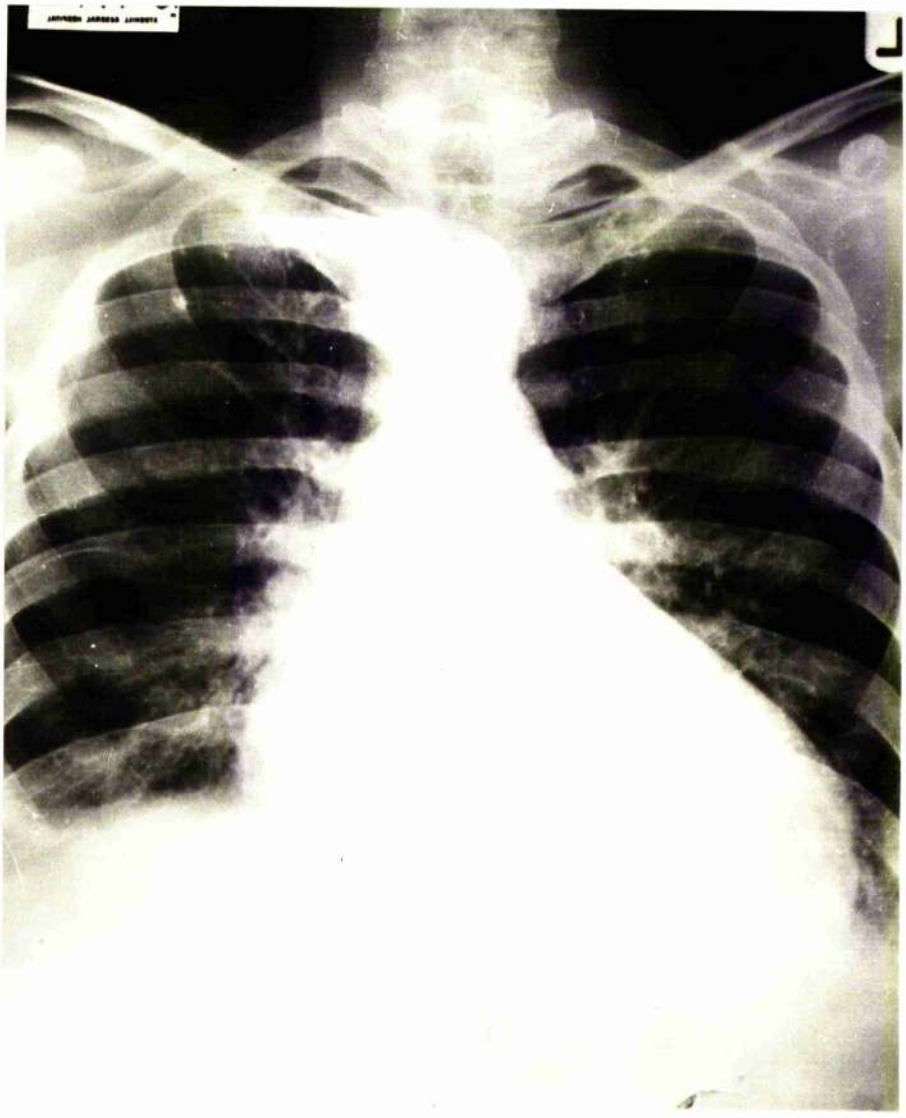


FIG. 9,5 CASE 15 - See Text

TABLE 9,8 10 PATIENTS AWAITING RDT
(Cuesta et al., unpublished observations)

	PCV %	Mean BP mm Hg	Total body water % of predicted	Cardiac output l/min	Stroke volume ml
Range	16 - 28	80 - 175	100 - 131	5·3 - 13·4	61 - 165
Mean	21·4	114·4	116·5	8·0	105·5
SD	4·5	26·0	9·7	2·9	29·7

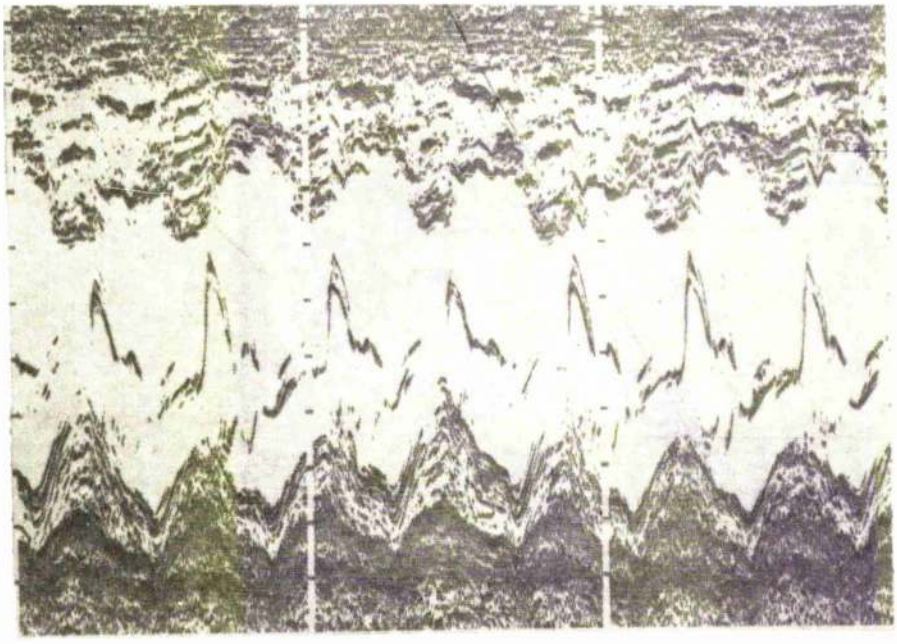


FIG. 9,6 CASE 4 - AFTER 12 MONTHS ON RDT

CHAPTER 10

A ROUTINE SERVICE IN CARDIAC ULTRASOUND

A routine echocardiographic service has been established in the Western Infirmary, Glasgow. The workload is shared between a cardiologist and a radiologist. There is nursing and secretarial assistance but no technicians are employed. On average, 5 patients are examined at each of 2 weekly sessions.

A limited evaluation of the service has been attempted. Technically satisfactory echograms and adequate clinical records were available in 201 patients examined between April 1971 and August 1973: the contribution made by ultrasound to their management has been investigated.

There were 6 main reasons for referral:-

1) To assess mitral stenosis
2) To assess mixed mitral valve disease
3) To exclude underlying mitral stenosis in patients with atrial fibrillation or unexplained cardiac decompensation
4) To assess the mitral valve in dominant aortic valve disease
5) To investigate an apical systolic murmur
6) To identify a pericardial effusion
7) Others

1) Mitral stenosis - 69 patients

a) 45 patients who subsequently underwent mitral valvotomy

The 37 patients discussed in Chap, 5 are included.

9 patients under my personal supervision and 5 others were not catheterised. In selecting them for operation, the echocardiographic findings were considered.

The remaining 31 patients were catheterised. The procedure precipitated atrial fibrillation in 3 instances and, given that echocardiography can differentiate adequately between a valve suitable for a closed valvotomy and one requiring an open procedure (Chap. 5), catheterisation added little to the echocardiographic/...

echocardiographic findings. Moreover, with the exception of one case where due to a measurement error, the severity of mitral stenosis was underestimated by ultrasound, the catheter and echocardiographic assessments of stenosis were in accord.

b) 9 patients with a history of previous mitral valvotomy

These patients were referred because of increasing symptomatology. The mitral echogram demonstrated a rigid valve in 3 cases and a pliable valve with a diastolic closure rate greater than 30 mm/sec. in the remainder. It was thus possible to conclude in each instance that further mitral valvotomy was not indicated. 3 patients were subsequently catheterised and the echocardiographic diagnosis of mild stenosis was confirmed in each.

c) 8 patients with established mitral stenosis and symptoms disproportionate to their signs.

These patients were thought clinically to have a respiratory or functional component to their symptoms. In each instance the mitral echogram demonstrated mild mitral stenosis. Thus cardiac catheterisation which in several instances was clearly undesirable, was avoided.

d) 7 patients with signs of mitral stenosis, presenting for the first time

The echogram established the diagnosis in each case. On echocardiographic grounds, the lesion was judged to be no more than moderate in 6 instances, in 3 of which no further investigation was thought necessary. 4 patients were catheterised and despite unequivocal echocardiographic evidence of tight mitral stenosis in one case, none was referred for operation.

2) Mixed mitral valve disease - 12 patients

In the period under review only 12 patients judged to have more than trivial mitral regurgitation were examined and 10 had an additional haemodynamic study. Echocardiography demonstrated a rigid calcified valve in 8 patients who proceeded to mitral valve replacement and a pliable valve with mild obstruction in/....

in 4 patients, none of whom was operated.

3) Exclusion of mitral stenosis - 27 patients

a) 3 patients with thyrotoxicosis and atrial fibrillation

The mitral echogram was normal in 2 cases. The third, who reverted to sinus rhythm, had been catheterised and a diagnosis of significant mitral stenosis had been made. The echocardiogram (Fig. 10,4) excluded significant mitral stenosis but demonstrated an unsuspected pericardial effusion which was subsequently tapped. The patient was subjected to further haemodynamic investigation which confirmed the ultrasonic findings.

b) 4 patients with lone atrial fibrillation

These patients were judged clinically to have lone atrial fibrillation. Their mitral echograms were normal and catheterisation to exclude underlying mitral stenosis was thus unnecessary.

c) 3 patients with evidence suggestive of pulmonary thrombo-embolic disease

A diagnosis of pulmonary thrombo-embolic disease was made at cardiac catheterisation in 2 patients who had no murmurs and who were in sinus rhythm. Echocardiography demonstrated tight stenosis of a pliable valve in one of them (Fig. 10,1): a further haemodynamic study which was complicated by the onset of atrial fibrillation, confirmed the finding.

The third patient was in atrial fibrillation and no further investigation was undertaken after a normal mitral echogram had been recorded.

d) 7 patients with chronic obstructive airways disease

These patients had clinical and laboratory evidence of airways obstruction. Despite the absence of a murmur, underlying mitral stenosis was suspected because of apparently disproportionate cardiac decompensation. 4 were in sinus rhythm and 3 in atrial fibrillation. The Mitral echogram was normal in 6 and stenotic in one patient. In the 3 patients subsequently catheterised, the echocardiographic findings were confirmed.

e) 4 patients with atrial fibrillation and unexplained cardiac decompensation

2/...

2 patients were shown by echocardiography to have mitral stenosis and one, after haemodynamic investigation, underwent a mitral valvotomy (Fig. 5,14b) In 2 instances the mitral echogram indicated that the valve was non-rheumatic.

f) 6 patients with atypical murmurs

In 5 patients including one in whom a diagnosis of mitral stenosis had been made at cardiac catheterisation 10 years previously, the mitral echogram was normal and further investigation was not undertaken. The sixth patient who had undergone multiple haemodynamic studies, none of which had excluded mitral stenosis, had a non-rheumatic echogram (Fig. 10,2): a diagnosis of mitral regurgitation was suspected but could not be confirmed.

4) The mitral valve in dominant aortic valve disease - 54 patients

See Chap. 6.

5) Apical systolic murmurs - 20 patients

a) 11 patients with suspected hypertrophic obstructive cardiomyopathy

The clinical suspicion was confirmed by typical echogram in 2 instances. The diagnosis was unequivocally excluded by ultrasound in 4 patients but in the remaining 5, the echograms proved difficult to interpret. Thus, Fig. 10,6 is from a patient with chronic renal failure and could be interpreted as showing the features of obstructive cardiomyopathy but echograms from the same patient on subsequent occasions were normal. The finding is unexplained but similar appearances have been recorded in a number of patients, particularly when the ultrasonic beam is directed towards the region of the posterior papillary muscle.

One patient (Fig. 10,5), whose obstruction had been relieved surgically, was examined 5 years post operatively.

b) 9 patients with possible sub-valvar mitral regurgitation

In 6 instances the mitral echogram appeared normal but it should be noted that at the time the examinations were made the importance of attempting to demonstrate cusp prolapse was not appreciated. Further, the relatively low diastolic/...

diastolic closure rates observed in these patients may simply reflect failure to record the full amplitude of the mitral echogram.

In 3 instances an increased diastolic closure rate and/or diastolic oscillation of the echogram (Chap. 7) were demonstrated and a diagnosis of mitral regurgitation was made. Fig. 10,3 is the echogram from a patient who was found at operation to have a "floppy" mitral valve, and Fig. 8,8 is from a patient with systemic hypertension and mitral regurgitation of acute onset.

6) Pericardial effusion - 12 patients

12 patients with chronic renal failure were referred because of increasing cardiomegaly. An echocardiographic diagnosis of pericardial effusion was made in 6 instances.

7) Others - 7 patients

3 patients, later operated, had echocardiographic features consistent with an atrial septal defect (Fig. 8,4).

4 patients were referred for examination of the tricuspid valve: the structure was visualised in only one of these (Fig. 10,7) and was apparently normal. Tricuspid echograms were also recorded on occasion in the dilated right ventricles of rheumatic mitral valve disease (Fig. 5,14) and atrial septal defect (Fig. 10,8).

This analysis has demonstrated that because clinicians are often reluctant to place reliance on echocardiographic assessment of the rheumatic mitral valves, many patients undergo unnecessary haemodynamic investigation. It has proved the value of echocardiography in the assessment of the mitral valve in aortic valve disease and in their investigation of other patients in whom mitral stenosis cannot be excluded on clinical grounds: the high yield of "silent" mitral stenosis is impressive. It has shown that ultrasound is often unsatisfactory in assessment of the non-rheumatic valve and may at times be unreliable in the identification of hypertrophic obstructive cardiomyopathy. Since these patients were studied the ultrasonic assessment of/...

of left ventricular function has been developed: this will obviously provide further valuable information.

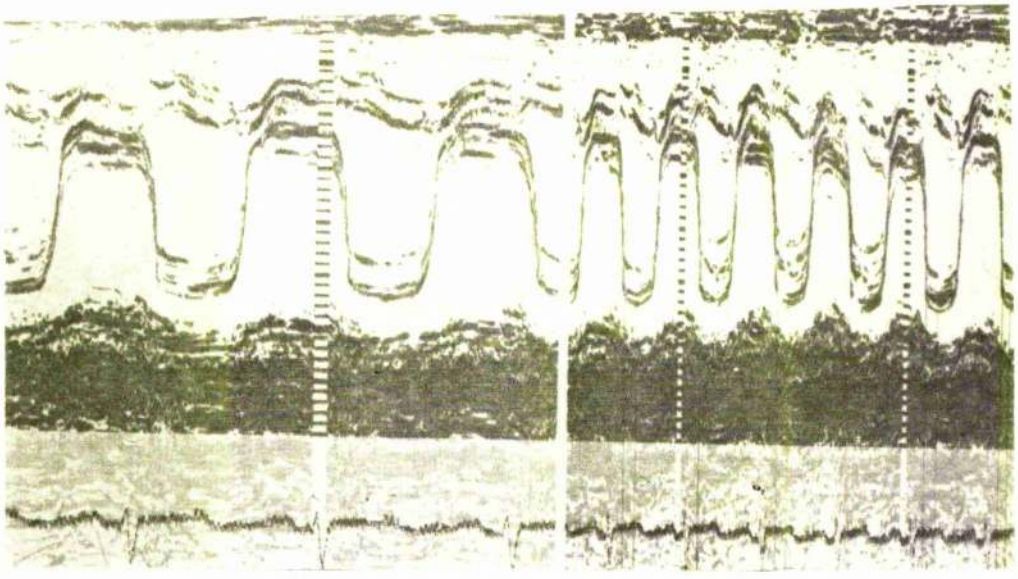


FIG. 10,1 "SILENT" MITRAL STENOSIS

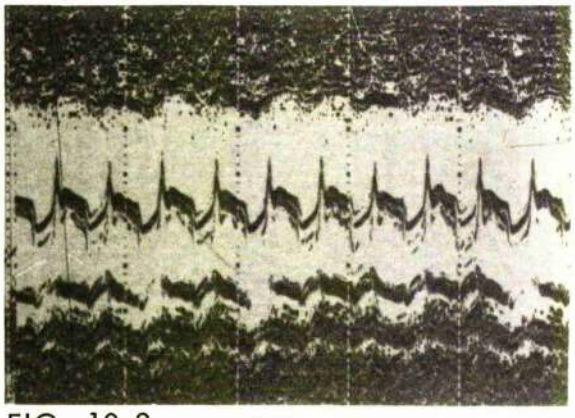


FIG. 10,2 SEE TEXT

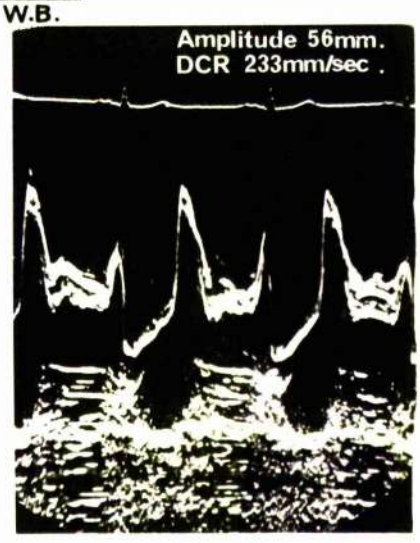


FIG. 10,3 "FLOPPY" MITRAL VALVE

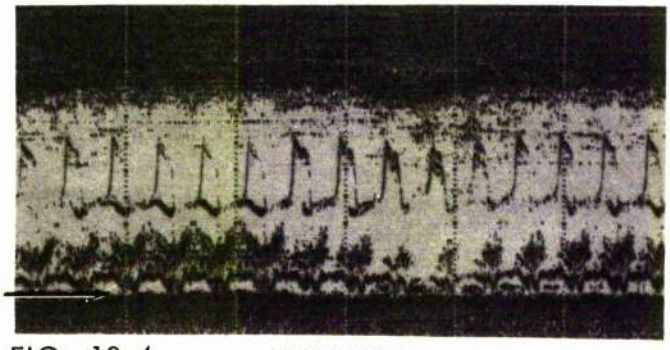


FIG. 10,4 SEE TEXT

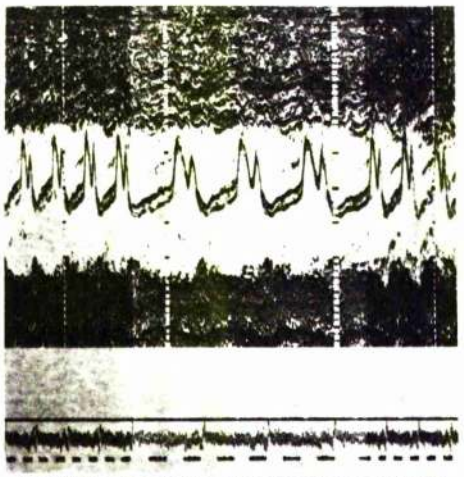


FIG. 10,5 HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY POST-OPERATIVE

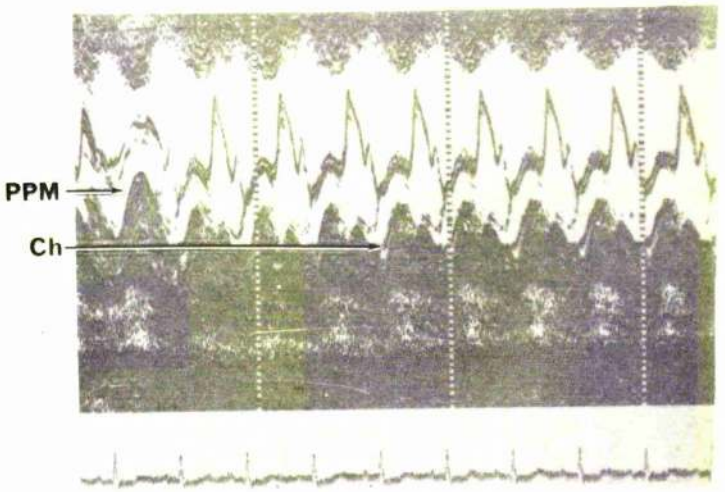


FIG. 10,6 SEE TEXT

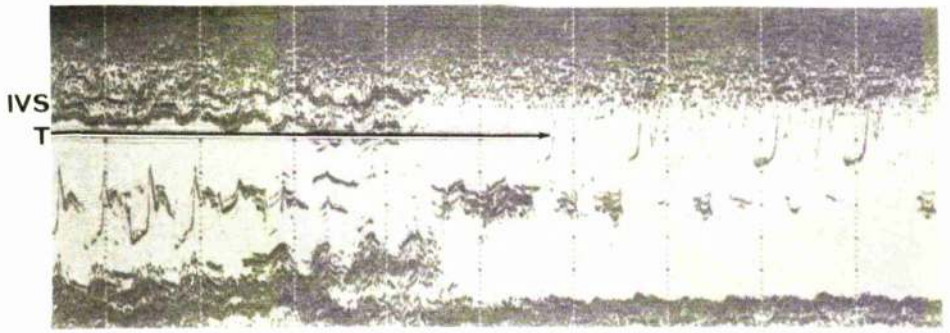


FIG. 10,7 TRICUSPID ECHOGRAM - See Text

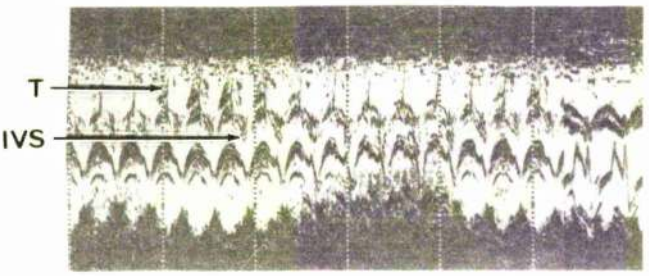


FIG. 10,8 TRICUSPID ECHOGRAM IN ATRIAL SEPTAL DEFECT

APPENDIX

	Amplitude mm.	DCR mm/sec	PWDV mm/sec	EDV ml.	ESV ml.	SV ml	EF
ormal	22.5	152	97	53	6	46	0.88
n = 13)	24.5	188	116	136	15	121	0.89
	27.5	200	125	136	33	103	0.76
	28		156	83	21	62	0.74
	28	139	83	146	38	107	0.74
	26.5	144	149	141	42	99	0.70
	31.5	167	100	85	18	68	0.79
	39	181	118	108	22	86	0.79
	30	203	109	115	17	98	0.85
	26	139	76	125	23	102	0.81
	28			139	42	97	0.76
	33	158	147	62	9	53	0.86
	25			93	16	77	0.83
ean	28.4	167	114	109	23	86	0.80
SD	4.3	25	28	31	12	23	0.07
R	21.5	290	167	422	131	291	0.69
n = 8)	24		107	250	94	156	0.62
	29		190	195	33	162	0.83
	17		90	336	129	207	0.62
	19.5		112	216	77	139	0.64
	26.5	154		216	37	179	0.88
	21.5		133	288	112	176	0.61
	31	300	136	381	244	143	0.37
ean	21.1	248	134	288	107	182	0.66
D	9.1	82	35	84	67	49	0.16
	<0.05	>0.10	>0.10	<0.001	<0.005	<0.001	<0.05
on-rheumatic	22	400	154	251	140	111	0.44
R	16.5	250	128	469	208	262	0.56
n = 10)	19.5			189	107	83	0.44
	21.5			143	89	54	0.38
	28			272	95	177	0.65
	27.5			200	137	64	0.32
	22.5			402	156	246	0.61
	32.5		216	166	68	98	0.59

cont/...

	Amplitude mm.	DCR mm/sec	PWDV mm/sec	EDV ml.	ESV ml.	SV ml	EF
Non-rheumatic	25	288	152	179	41	138	0.77
MR	35	500	62	184	64	120	0.65
Mean	25	356	142	246	111	135	0.54
SD	5.8	113	56	108	50	72	0.14
p	> 0.10	< 0.01	> 0.10	< 0.005	< 0.001	< 0.10	< 0.001
MS	29	14	54	56	15	42	0.75
(n = 7)	34	26	27	76	26	56	0.74
	26	12	24	84	24	60	0.72
	25	15	40	69	24	45	0.45
	28	40	24	53	14	39	0.74
	26	7		97	13	84	0.87
	24	12		141	54	87	0.62
Mean	27.4	19	34	82	23	59	0.70
SD	3.4	12	13	30	14	20	0.13
p	> 0.10	< 0.001	< 0.001	< 0.10	> 0.10	< 0.02	< 0.10
MDT	27	157	120	186	22	165	0.88
(n = 15)	22.5	227	190	298	138	159	0.53
	33.5	182	106	216	91	125	0.58
	24.5	162		145	31	114	0.79
	33		145	216	43	173	0.80
	32	178	103	193	52	142	0.73
	28.5	177		118	20	98	0.83
	31.5		142	178	41	136	0.79
	37	232	76	204	54	150	0.74
	29	211	80	145	22	123	0.85
	25	188		94	55	39	0.42
	25		80	166	55	112	0.67
	28		75	197	35	162	0.82
	25	236	213	369	319	50	0.14
	26	209	94	145	23	122	0.84
Mean	28.5	196	119	191	67	125	0.69
SD	4.1	28	46	69	76	39	0.20
	> 0.10	< 0.02	> 0.10	< 0.001	< 0.05	< 0.005	< 0.10

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