Non Invasive Assesment of 42 Cases of Dilated Cardiomyopathy (Idiopathic)

By

Abbasi, A. Karim*; Sangi, S.A.**; Soomro, K.** and Soomro, T.**

Introduction

The diagnosis of Cardiovascular disease by history, examination of pulse wave, inspection, palpation, apical cardiac movements, ausculation of the heart sounds and murmurs, is supplemented by external graphic recordings as an improvement since long. Systolic Time Intervals (STI's) and Diastolic Time Intervals (DTI's) are assessment techniques for the Cardiace performance, which is influenced by the end diastolic fibre length, mean aortic pressure, myocardial contractility, heart rate and contraction synergy.

The use of external non body penetrating techniques, for the confirmation of clinical findings, diagnosis and therapeutic applications are of great value. By these methods patients who are seriously sick and can not stand invasive measures, can be assessed by the bed side. Patients liable to undergo surgery, could be assessed pre-operatively, for the suitability and indications for operation; also post operatively, for the results achieved, and for the therapeutic measures to be applied. The interaction of certain drugs on the heart could also be assessed. The objective of this study was to find out in details changes, in various phases of cardiac cycle, Phonocardiogram (PCG) and Apexcardiogram (ACG) and carotid pulse in cases of idiopathic dilated cardiomyopathy.

Material & Method

Patient Selection :

Heart muscle disease has divergent etiology of known and unknown origin. Patients with cardiomegaly, with no evidence of Rheumatic heart disease, coronary heart disease, Hypertension, Left to right shunts and any other congenital or acquired cardiac defects, were subjected to ECG, X-Ray chest & Echographic recording for exclusion of secondary etiological factors. These cases were labelled as idiopathic dilated cardiomyopathy for further assessment of the cardiac function.

50 patients with age variation of 3—50 years were included in this study. after final exclusion completed Out of these, 8 patients were dropped from the study due to various conduction defects (Table-1). All these patients were in Cardiac failure and all of these were on digoxin, diuretic and preload reducing agents.

* Professor of Cardiology.
** Registrars in Cardiology, Liaquat Medical College, Hyderabad.

GENERAL INFORMATION
Table—I

TOTAL CASES IN STUDY	:	50
EXCLUDED	:	8
LBBB	:	4
RBBB	:	1
WPW	:	3 { TYPE A—1 CASE / TYPE B—2 CASES
NO. OF CASES PRESENTED	:	42
IST DEGREE A.V BLOCK	:	24 (57%)
ATRIAL FIBRILLATION	:	10 (24%)
R.S.R WITHOUT BLOCK	:	8 (19%)

Equipment and Method Used :

Mingograph 81 — 8 channel recorder with, multiple band pass filters, for the recording of heart sounds & murmurs and special apex & carotid pulse pick ups, with fixed Lead-II ECG, was used.

All the recordings were done in held end expiration between 8—10 am and the patients were put in semi left recumbent position. The apex was localized by inspection, palpation and the apex pick up EMT—510 Elema type was applied to the patient at maximum impulse and held in position by the rubber strap, with special attention to prevent air leaks under the pick up, and to maintain constant touch with the body surface. Phono pick up Elema EM 25.C was applied in the 4th left intercostal space in the mid precordial region. The carotid pick up was applied above the clavicle at the point of visible carotid impulse, and was held in position by the hand. Lead—II ECG was also applied as referral tracing. The recordings were done with gains adjusted equally on the 8—channels. The heart sounds were recorded initially, then apex and carotid waves were
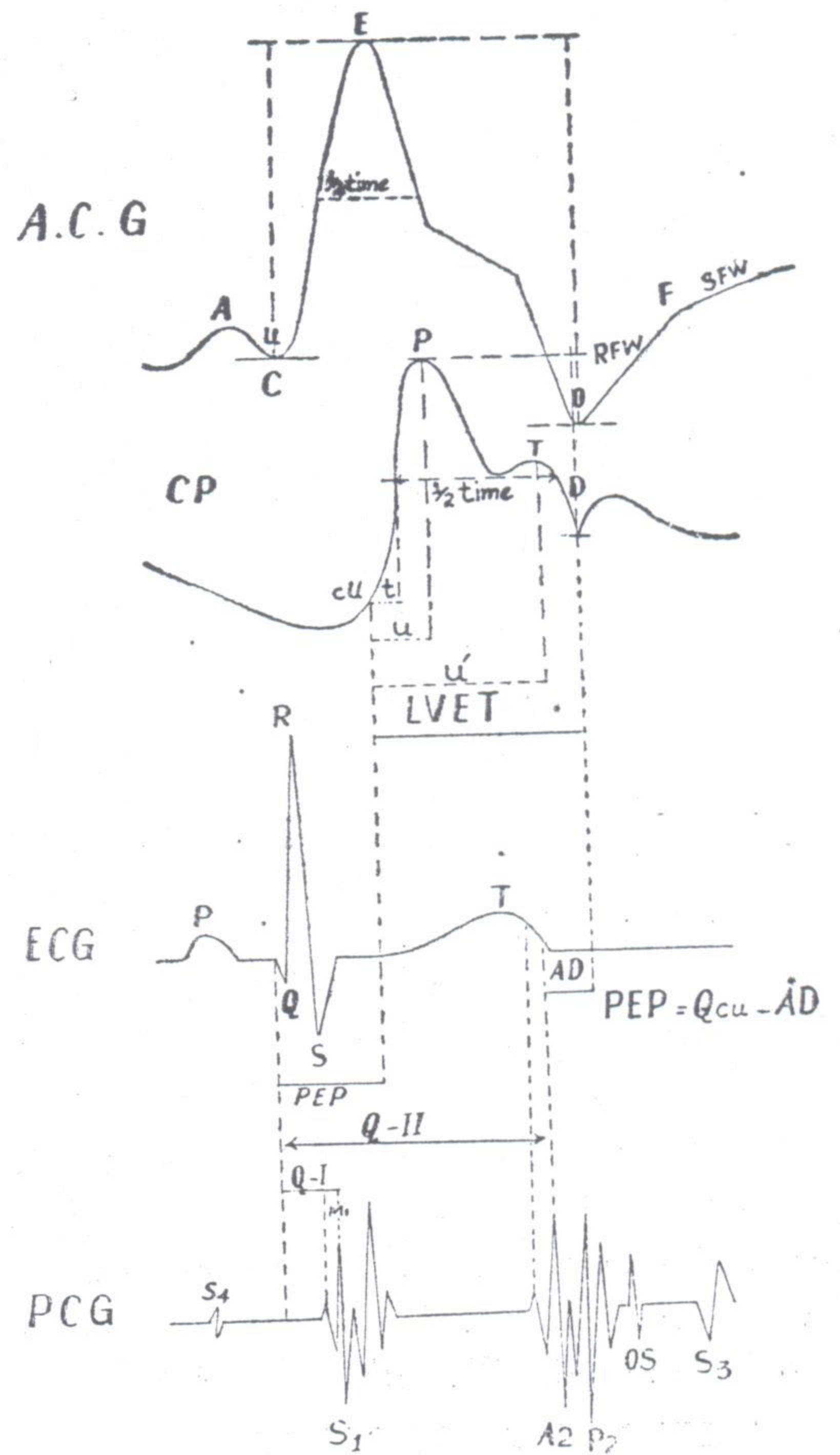

Fig. 1 : Shows correlation of ACG, CP, ECG & PCG.

added to heart sounds. All the recordings were done with the paper speed of 100 mm/sec and all the interpretations done in the frequency range of 200 cycles/second.

The measurements of STIs, DTIs, apex and carotid wave variables were done according to the methods published by Weissler and Ben-chimol (Figs. 1, 2 & 3). All the variables values obtained on 5 consecutive cardiac cycles for each patient were adjusted to their means. The tracings were re examined by two co-workers in study on the above pattern and average of these 3 readings was accepted as final mean value for the individual patient The abbreviations used in the study are the standard as used by Weissler et al.
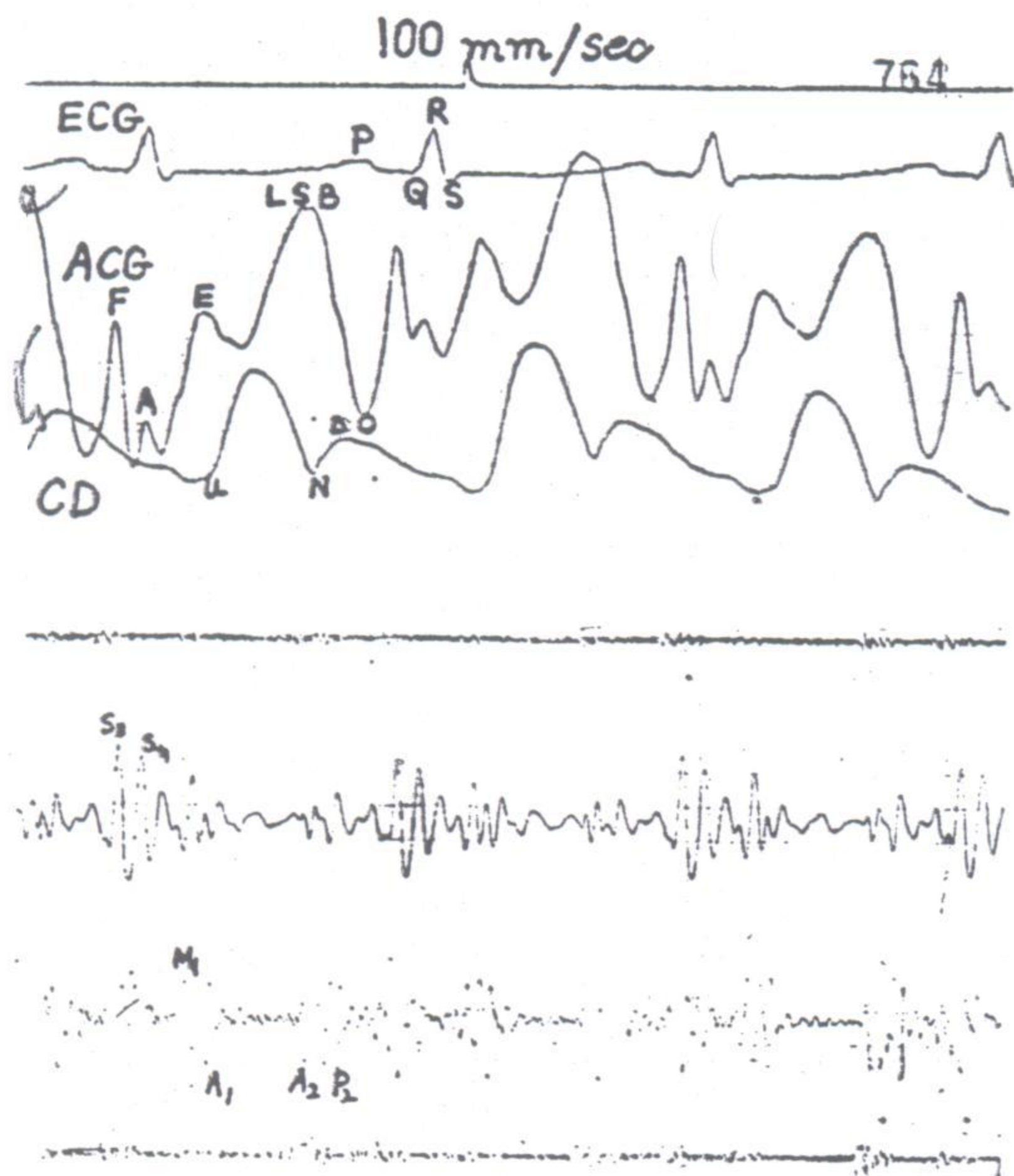

Fig. 2:

Shows lateral correlation of ECG, ACG, CD and PCG. It also shows prominant F-waves, LSB, pulsus & apex alternans concordant.
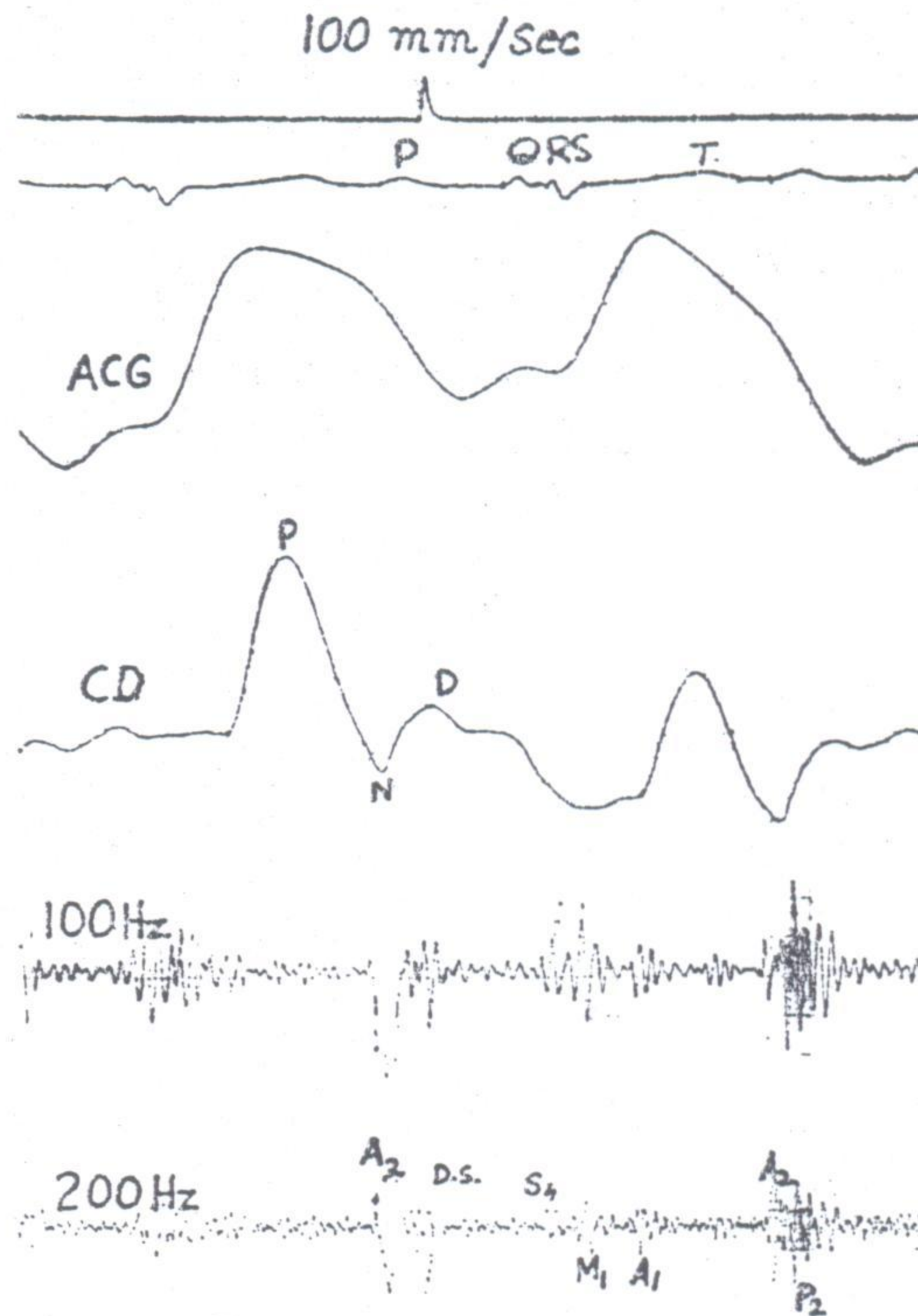

Fig. 3:

Shows ECG, ACG, CD and phonocardio-graphic recording with pulsus & apex alternans discordant.

Weissler Regression Equation for the heart rate and sex was used for each individual, and thus predicted values were drawn for the major phases of cardiac cycle. Every possible effort was done to get good quality tracings for interpretation and inclusion in the study Defective tracings were excluded. For the comparison of the observed values 40 normal subjects of nearly same age group and sex distribution were used where ever predicted values could not be applied Unpublished data). Ten patients had AF which made it

difficult to read all the phases of cardiac cycles in 42 cases, thus each phase of cardiac cycle was read in vertical manner in relation to RR interval for the particular number of cases, which resulted in inequities in the horizontal results. The cycle lengths in this paper thus have to be read vertically with reference to number of cases against each division. All the cycle length values are expressed in msec & ratios in %.

Results

General Information :

Initially 50 cases were included in the study, out of which 8 had to be dropped because of intraventricular conduction defects (Table—I). 1st degree AV block was present in 24 cases (57%), AF in 10 cases and remaining 8 had regular sinus rhythm, without any conduction defect.

The age varied from 3—50 years with the mean of 24 with SD of 14.3. Male to female ratio was 3:1 and mean BP=87 mm Hg (Table-II).

ECG lead-II for all these patients showed mean QRS=79, QR=36, PR=158, RR=542m sec & heart rate of 115/min (Table-III).

GENERAL INFORMATION

Table—II

Variable.	No. of cases	Min.	Max.	Mean	SD
AGE (YEARS)	42	3	50	24	14.3
SEX	42	—	—	—	—
BSA (m2)	42	0.73	1.88	1.47	0.35
BP (mmHg)	42	73	96	87	6

ECG LEAD—II INFORMATION.

Table—III

Variable.	No. of cases	Min.	Max.	Mean	SD
QRS msec	42	60	100	79	12
QR msec	42	25	45	36	5
PR msec	32	130	190	158	21
RR msec	42	410	850	542	127
HR/min:	42	71	146	115	21

Note: All measurements are in msec.

STIs: _____

STIs (Table-IV) deal with various phases of cardiac cycle in systole. Electro-mechanical delay (QC) was found to be unchanged, with increase in Pre-isovolumic contraction time (C-M1) mean 49 SD±10 msec The electroacoustic interval (Q-M1) was found to be increased by 92% in all the cases with mean of 91 SD±10 msec. The pressure elevation time (M1-E mean 34

SYSTOLIC TIME INTERVALS

Table—IV

Variable.		No. of cases	Min.	Max.	Mean	SD
Q—C msec		42	30	60	42	9
C—M1 msec		40	25	60	49	10
*Q—M1 msec	PRED.	42	39	61	47	6
	MEAS.	42	70	110	91	10
M1—E msec		40	15	70	34	13
C—E msec		42	55	140	83	19
*PEP msec	PRED.	42	75	103	86	8
	MEAS.	42	100	180	126	18
*Q—A2 msec	PRED.	42	257	397	308	40
	MEAS.	42	260	400	309	40
C-M1/M1-E %		42	100	267	159	55
*LVET msec	PRED.	42	184	299	223	31
	MEAS.	42	150	270	197	38
*PEP/LVET%	PRED.	42	35	41	39	1.66
	MEAS.	42	44	87	66	14
*EF 1.125-(1.25XPEP/LVET) %	PRED.	42	61	68	64	2
	MEAS.	36	16	57	36	13
*M1—A2 msec	PRED.	42	227	328	258	30
	MEAS.	42	175	310	231	43
C—A2 msec.		42	210	360	280	47

*P value = < 0.05.

SD±13), was found to be less than 30 msec in 24% cases with 1st degree AV block, and the reduction in M1-E was not parallel to the increase in C-M1.

True isovolumic contraction time (C-E mean 83 SD±19) was increased. 10% of cases had C-E interval more than 99 msec.

Pre ejection period (PEP mean 126 SD±18) was increased in all the cases by 47%. The electromechanical systole (Q-A2 mean 309 SD±40) remained unaltered. The IVCT index (C-M1/M1-E mean 159 SD±55) was increased in all the cases. L.V ejection time (LVET mean 197 SD±38) was decreased by 11%. The PEP/LVET ratio (mean 66 SD±14) was increased by 70%. Ejection fraction (EF mean 36 SD±13) decreased by 44%. EF was calculated by the formula 1.125—(1.25 X PEP/LVET)%.

Mechanical systole (M1—A2 mean 231 SD±43) was decreased by 11%. True mechanical systole (C—A2 mean 280 SD±47) was found to be increased.

DTIs :

(Table—V): The diastole (A2—Mi mean 278 SD±26) was found to be increased by 11%. Isovolumic relaxation time (A2—O mean 86 SD±13), and A2—F (mean 132 ED±13) remained within low normal range. The OF/EO ratio (mean 27 SD±18) was increased. The CM1—OF interval (mean 8±16 SD) was found to be negative in 33% cases with 1st degree AV block. The remaining had positive values. Slow filling wave (SFW mean 36±13 SD) showed disproportionate decrease with reference to the heart rate. The OF/SFW (mean 133±46 SD) was found to

be increased in all cases. The S—a interval (mean 131 SD±26); maintains distinct time relationship (Hartman & Snellen, 1960), and was found to be more than 149 msec in 29% of cases with 1st degree heart block.

The ACG a-wave breadth (mean 66 SD±21) was found to be within normal range. 16% of the cases with 1st degree AV block had 'a' wave breadth more than 66 msec. a/ED ratio (mean 18 SD±9), was increased and 19% cases with 1st degree AV block had a /EO ratio more than 14%.

ACG :

(Table—VI) The E-point of ACG which coincides with the maximum dp/dt of L.V (Benchimol et al), was found to be sharp in most of the cases (86%), followed by sustained systolic wave (86%) with LSB (100%) and prominant F wave (95%). The change of slope of systolic wave (B—A2) % was found to be early in all the cases, with CE/ED ratio (mean 62 SD±11) reduced. The ½ time RR% (mean 42 SD±13) was found to be increased. 19% patients with prolonged PR interval had ½ time interval more than 35%.

Carotid :

(Table—VII) The ratio of, downward displacement to the total amptitude of systolic wave (dt/dp mean 71±15 SD) was increased. The amplitude of the dicrotic wave with reference to the total displacement of systolic wave (D/dp mean 29 SD±8) was increased in all the cases. The ½ time RR% (mean 29 SD±4) remained unaltered. The rate of rise of carotid pulse wave (mean 726 SD±157) remained with in normal limits. Only 10% of cases had a rate of rise of more than 999 mm/Sec, but still within normal range.

DIASTOLIC TIME INTERVALS.

Table—V

Variable		No. of cases	Min.	Max.	Mean	SD
A2—M1*	PRED.	36	183	303	249	40
msec	MEAS.	36	240	330	278	26
A2—0 msec.		42	70	110	86	13
A2—F msec.		42	110	160	132	13
OF BREADTH msec.		42	30	60	44	9
OF/EO %		42	14	77	27	18
CM1—OF msec.		42	—15	40	8	16
SFW msec.		32	20	60	36	13
OF/SFW %		32	70	200	133	46
P—a msec.		32	90	170	131	26
a—Q msec.		32	—10	55	20	18
a-WAVE BREADTH msec.		32	40	100	66	21
a/EO %		32	4	33	18	9
A2—S4 msec.		34	150	250	197	25
S4—M1 msec.		36	50	100	79	15
P—S4 msec.		16	130	210	161	32

* P value = <0.001.

ACG

Table—VI

Variable.	No. of cases	Min.	Max.	Mean	SD
$\dfrac{B-A2}{M1-B}$ %	42	130	633	256	124
$\dfrac{CE\ AMP}{E-O}$ %	30	48	81	62	11
$\frac{1}{2}$ TIME % RR	32	21	58	42	13

L.S.B	POSITIVE IN	100%
	SUSTAINED IN	86%
	EARLY COLLAPSE IN	14%
PROMINANT F WAVE IN		95%

CAROTID

Table—VII

Variable.		No. of cases	Min.	Max.	Mean	SD
dt/dp %	..	36	43	100	71	15
D/dp %	..	30	19	50	29	8
$\frac{1}{2}$ TIME % RR	..	36	21	37	29	4
RATE OF RISE mmHg/sec	..	40	500	1250	726	157

Discussion

Wiggers delieniated various phases of the cardiac cycle long ago, and it is since then that the systolic and diastolic time intervals as drawn by using ECG, PCG, ACG and carotid pulse wave tracings, have been used for the assessment of ventricular performance. STIs refer to the temporal sequence of venticular events in systole, and the DTIs refer to the dynamic changes during diastole.

Cardiomyopathies with divergent etiologies, known or unknown, are more frequently diagnosed than previously, due to better diagnostic facilities.

STIs :

The systolic period of the left ventricle consists of, electromechanical systole (Q-A2), LVET and PEP as the major phases. These phases are further sub-divided into smaller intervals (Wiggers). In our study of 42 cases of cardiomyopathy of unknown origin, the electromechanical systole remained unaltered, where—as PEP, PEP/LVET ratio showed an increase, and the LVET was decreased. These changes observed in our study are consistent with previous reports (Weissler et al 1968). The electro acoustic interval (Q-M1) was increased significantly in all our cases. The extent of the prolongation is affected by multiple factors like the QRS interval, variation of the electromechanical delay (QC interval) from individual to individual, length of the preceding cycle, and the individual variation of the rate of pressure rise in left ventricle.

In cardiomyopathy the myocardial contractility and relaxibility are decreased, and the rate of pressure rise in L.V is also decreased. This causes prolongation of the pre-isovolumic contraction time (C—M1), and delayed closure of the mitral valve. The iso-volumic-contraction time (M1—E) is an index of the ventricular contractility. During this period the ventricle builds up pressure without undergoing any change in its volume, while the mitral and aortic valves remain closed. The only change that occurs during this interval is in the length and tension of the myocardial fibre. Digitalis increases this interval by improving myocardial contractility (Weisser et al 1968). In our study delayed mitral valve closure appears to have contributed to a minor reduction in the M1—E interval, because of the fact that all our patients were on digoxin. The dilated L.V. because of ineffective contractility, led to the prolongation of the pre-isovolumic-contraction time, which reduces M1—E due to delayed mitral valve closure. However the degree of reduction in M1—E is not parallel to the increase in C-M1 interval. The IVCT index (C-M1/ M1-E) appears to be a better index of the contractility of L.V. (Willems and Oreshkov et al. 1971). Patients in our study all showed an increase of the IVCT index. An overall increase in the true IVCT (C-E) was also noted.

The duration of PEP comprising QC, CM1, and M1-E reflects ventricular contractility, which is directly related to the end diastolic fibre length, was increased. The group of patients in our study had dilated left ventricle. The LVET was decreased in all the cases because of the poor inotropic state of L.V. myocardium. Weissler et al in 1969 had established that the PEP/LVET ratio varies within a narrow range in the normals. It serves as a useful index for the judgement of L.V. performance. This ratio is increased in all our

cases, because of diminished myocardial compliance and a prolonged P-R interval. Digitalis diminishes PEP/LVET ratio (Weissler et al 1969). The mean values thus presented here may not reflect the real increase in PEP/LVET ratio.

The ejection fraction (EF) related to volume changes in the L.V. during systole and diastole was reduced in all our cases due to poor compliance of the L.V.

The true IVCT (C-E) showed an increase over the normal values of Benchimol (74 msec) and our laboratory values of 70 msec. The mild increase in C-A2 interval appears to be due to the prolongation of C-M1 interval.

The mechanical systole (M1-A2) dependant on pressure elevation time and L.V. ejection time in our study was decreased by 11% which is the reduction seen in LVET.

DTIs :

Various phases of cardiac diastole as drawn by the use of ECG, PCG and ACG are, to understand the hemodynamic changes that occur during diastole (Willems, Jos. L., 1971).

A2-0 interval is related to the rate of fall of pressure in L.V. It depends on aortic pressure at the time of closure of aortic valve, myocardial relaxation and L.A. pressure at the time of mitral valve closure (Arevalo, Federico, 1969). Cardiomyopathies with diminished contractility and relaxability, decreased stroke output, have low aortic valve closing pressures and high L.A. pressure, leading to decreased A2-0 interval. In our study A2-0 interval was seen in low normal range. In normal individuals LVET and A2-0 intervals are inversly related;

but in dilated cardiomyopathy they are directly related; because of decreased contractility and relaxability.

The rapid filling phase of the L.V. starts after mitral valve opens and the height and breadth of OF depends on, the amount and velocity of the flow across the mitral valve, L.A. pressure and the impact this flow creates on the L.V. wall. In cardiomyopathies with increased L.A. pressure and decreased L.V. pressure, OF/EO is more than the normal value of 15%. After the rapid phase of ventricular filling there is reduced filling of L.V.(SFW). The clinical importance of this phase is still to be known, however it varies disproportionately to the heart rate. In a normal study conducted by Benchimol et al, OF/SFW is in the ratio of 1:3, but in our study this ratio is increased. This indicates the prolongation of the slow phase of ventricular filling in cardiomyopathies.

The a-wave of ACG has distinct time relationship with the P-wave of ECG; but in our study it is seen to be increased in 29% of the cases amongst the 57% cases of Ist degree AV block. (P-a more than 149 msec), and it was within a normal range in the remaining cases with AV block.

Bundle of His electrocardiography could not be done to confirm the site of block in AV junction and its relationship to above refered P-a interval. The late diastolic ventricular filling caused by the L.A. contraction causes 'a' wave in ACG. The height and breadth of this wave is the result of L.A pressure, L.A. contractibility and L.V compliance (Braunwald et al 1963, Wayne, Howard A. 1973). In our study

the height of 'a' wave was increased in the majority of cases. However some patients had 'a' wave height in the normal range. The breadth of 'a' wave mean values are within the normal range. a/EO ratio when increased more than 14%, is associated with increased LVEDP (Epstein, E.J. et al 1966). In our study some cases had a/EO ratio within the normal range, while others had a more than the normal range. The mean values were more than 14%. The low normal value of A2-O interval and the normal breadth of the 'a' wave in our study, was probably due to the fact that our patients were on digoxin and diuretic therapy. Digoxin prevents rise in a/EO and the breadth of 'a' wave, and improves the contractility of both the atria and the ventricle; and thus have contributed, alongwith other factors, in the low normal A2-O interval. A2-M1 interval in our study is increased by 11% with reference to predicted values, due to delayed mitral valve closure. CM1-OF interval was used by Oreshkov alongwith QM1-A2O interval to asses the severity of mitral stenosis. More positive values in both these were taken as indication of the advanced mitral valve narrowing, but in our study mean QM1-A2O interval and mean CM1-OF interval are increased, inspite of the fact that some of our patients had negative CM1-OF & QM1-A2O interval. This index seems to be more related to the volume flow through the mitral valve, than the size of mitral valve, and its utility to asses cardiomyopathy is poor.

ACG :

The graphic recording of low frequency vibration of the chest wall at the site of maximum apical impulse, reflects the dynamic changes in L.V (Willems, Jos. L., 1971). ACG reflects changes in L.V. without appreciable delay (Tavel et al 1968 & Manolas et al 1975). Large 'a' wave may even be absent or minimal inspite of raised LVEDP (Voigl & Friesenger 1970). a/EO 'ratio in normals is not more than 15% (Epstein et al 1968 & Benchimol & Diamond 1962); was found to be increased in majority of our patients. The L.V. systolic wave retracts after E-point which relates to maximum dp/dt of L.V. and opening of the aortic valve. The change of slope in normals is beyond mid systole. In our study the change of slope was found to be early in all the cases. The systolic wave was sustained in 86% and LSB was seen to be present in 100% cases. None of these patients had L.V. hypertrophy (Echo study), thus sustained systolic wave is due to increased wall stress in dilated and poorly compliant ventricles. The left sternal bulge (LSB) was reported by Benchimol & Diamond 1963 & Fowler 1968 in ischemic heart disease due to segmental involvement of the myocardium. The LSB seen in our study, in cases of cardiomyopathies may be the result of differential myocardial contractility and left ventricular forward reaction to posterior ejection into L.A. as reported by Mounsey, 1967 in cases of mitral regurgitation. The CE/EO ratio and $\frac{1}{2}$ time interval RR% are inversly related in our study of cardiomyopathy and both the ratios are dependant on myocardial fibre shortening. In ACG recordings in our study, apex alternans was seen in serious cases.

Carotid Pulse (CD) :

The carotid wave recording closely correlates with the aortic wave recording. The wave pattern depends on multiple factors, like heart rate, stroke-volume, L.V. outflow tract

anatomy, aortic valves, aortic ring, condition of the aorta, elasticity of the vessels and contractility of the myocardium. LVET is decreased in cardiac failure (Weissler et al, 1961).

Digitalis reduces LVET in normal and abnormal hearts (Weissler et al, 1966).

McKenzie in 1902 described carotid pulse by saying that decreased ventricular contractility is associated with decreased systolic wave amplitude, with very low notch and prominant dicrotic wave. In our study, we found single systolic wave with low notch and prominant dicrotic wave as also reported by Ewy, Gordon A. 1969. The ratios of the systolic downstroke to systolic upstroke of the carotid systolic wave and the height of dicrotic wave to systolic upstroke, is increased in all the cases, whereas ½ time RR% interval is within normal range, indicating none of the patients in study had valvular disease.

The initial rate of rise of carotid pulse is within low normal range and only 10% of cases had the rate more than 999 mm/Sec.

In our study we observed pulsus and apex alternans both concordant and discordant in seriously sick patients. The tall systolic wave in these cases with pulsus alternans is associated with low dicrotic wave; and low systolic waves are associated with high dicrotic wave.

The dicrotic wave caused by rebound of blood against the closed aortic valve is related to PR interval and force of ventricular con-traction.

The tall dicrotic wave indicates functional impairment of the myocardium.

Conclusion :

The study of STIs, DTIs, ACG & carotid pulse wave changes in primary myocardial disease, as done in this study, helps to under-stand changes in various phases of Cardiac cycle. The prolongation of PR interval does not affect all the phases of cardiac cycle. Only certain percentage of cases with prolonged PR interval affected various phases of cardiac cycle probably due to variable site of block in the A.V. junction. Since all the patients were on digitalis, diuretics and venodilator therapy, effects of digitalis are noted in certain phases of cardiac cycle. Volumenous work has been done in the past to find out reliable left ventricular performance indices, could be used as a specific, sensitive and reliable measure for the judgement of L.V. function. Many of them together can indicate altered L.V. function.

Summary

Over the past thirty years the new techniques of biotechnology have touched most of the recalcitrant problems in the cardiac diagnosis. Non-invasive assessment of 42 cases of Indiopathic Dilated Cardiomyopathy by using ECG, PCG, ACG & Carotid pulse wave, are presented here. The diagnosis of Idiopathic dilated cardiomyopathy was done by exclusion.

Endomyocardial biopsies and Angio studies of these cases were not be done.

The age varied from 3-50 yrs with mean of 24 ± 14.3 SD and male: female ratio was 3:1.

STIs show increase in CM1 (Mean—49 ± 10 SD),

QM1 (Mean—91 ± 10 SD), PEP/LVET ratio (Mean — 66 ± 14 SD), C-M1/M1-E% (Mean—159 ± 55 SD).
STIs shows decrease in M1-E (Mean 34 ± 13 SD), LVET (Mean—197 ± 38 SD), EF (Mean—36 ± 13 SD) and
M1—A2 (Mean—231 ± 43 SD) with no change in Q—A2 interval. DTIs show increase in A2-M1 (Mean—278 ± 26 SD), OF/EO ratio%- (Mean 27 ± 18 SD), CM1—OF (Mean 8 ± 16 SD), OF/SFW% (Mean—133 ± 46 SD) & a/EO%— (Mean—18 ± 9 SD).

ACG shows early change of slope, prominent F-wave, sharp E-point, LSB & increase in $\frac{1}{2}$ time RR%. Carotid pulse-wave shows prominent dicrotic wave, normal $\frac{1}{2}$ time RR% & lower normal rate of rise.

Abbreviations: 1. PCG—Phonocardiography
      3. STIs—Systolic Time Inter-
         vals
      2. ACG—Apex Cardiography
      4. DTIs—Diastolic   Time
           Intervals

References

1. Abbasi, A. Karim, Non invasive assessment of 100 Normal Males. Pakistan Heart Journal, Nov. 1983.

2. Aldo. A. Lusaida, The sounds of normal heart, 1973.

3. Aldo. A. Lusaida, From auscultation to phonocardiography, 1965.

4. Aranow, W.S, Isovolumic contraction & left ventricular ejection time, American J. Cardiol; 26:238, 1970.

5. Aubrey Leatham, Auscultation of heart and phono cardiography, 1975.

6. Arevalo et al: On the duration of isovolumic relaxation period in dog and man. Am. Heart J. 35:691, 1973.

7. Benchimol, A: A study of the period of isovolumic relaxation in normal subjects and in patients with heart diseases. Am. J. Cardiol; 19:169, 1967.

8. Benchimol, A: Non invasive techniques in cardiology for nurses and technicians, 1978.

9. Benchimol, A: Non invasive diagnostic techniques in cardiology, 1981.

10. Benchimol et al: The normal and abnormal apex cardiogram, Am. J. Cardiol. 12:368, 1963.

11. Braunwald et al: Time relationship of dynamic events in the cardiac chambers, pulmonary artery and aorta in man. Circ. Res. 4:100, 1956.

12. Braunwald et al: Assessment of cardiac function. Ann. Int. Med: 70:369, 1969.

13. Braunwald et al: Mechanism of the contraction of normal and failing heart. New Eng. J. Med. 1967.

14. Braunwald, The ventricular end diastolic pressure. Am. J. Med, 34:147, 1963.

15. Dean. T. Mason et al: Clinical determination of left ventricular contractility by hemodynamic and myocardial mechanics. Progress in Cardiology, 1972.

16. Elliot Rapaport et al: Cardiology Update, 1979.

17. Edward. K. Chung, Non invasive cardiac diagnosis, 1976.

18. Ewy et al: The dicrotic arterial pulse, Circ; 39:655, 1969.

19. Frank M.N. et al: Indirect measurement of isovolumic contraction time and tension period in normal subject. Am. J. Cardiol; 10; 800, 1962.

20. Garrard et al: Relationship of alterations in systolic time intervals to ejection fraction in patients with cardiac disease. Circulation; 42:455, 1970.

21. Golde et al: Systolic phases of the cardiac cycle in children. Circulation, 42:1029, 1970.

22. Goodwin J.F., The cardiomyopathies. Brit. Heart J. 34:545, 1972.

23. Gibson et al: The a wave of apex cardiogram and left ventricular diastolic stiffness. Circulation; 49:441, 1974.

24. Howard. H. Wayne, Non invasive techniques in cardiology. 1973.

25. Harris et al: Severe pulsus alternans associated with primary myocardial disease in children. Circulation; 34:948, 1966.

26. Harvey. W.P, Auscultatory findings in primary myocardial disease. Am. Heart J. 61:199. 1961.

27. Hudson. R.E, Cardiomyopathies: Order from Chaos. Am. Journal of Cardiol 25:70, 1970.

28. Irwin. R. Callen et al. Symposium on Non invasive cardiac diagnosis. Medical Clinics of North America; 64:1, 1980.

29. John. F. Goodwin, Future trends in Cardiomyopathy. Progress in Cardiology. 10, 1981.

30. Jules Constant, Beside cardiology, 1976.

31. Kumar, S. and Spodick, D.H: Study of the mechanical events of the L.V. by atraumatic techniques. American Heart Journal; 80:401, 1970.

32. Karnegis et al. The Q, inverval of the phono cardiogram. Am. J. of Cardiol 11:452, 1963.

33. Lewis, R.P; et al. Abnormal systolic time intervals with normal cardiac output in myocardial disease. Circulation, 42:1970 Abstract.

34. Lane et al. The apex cardiogram and myocardial asynergy. Circulation, 37:890, 1968.

35. Laglar et al, The apex cardiogram in the study of 20s interval, Brit. Heart. J. 25:246, 1963.

36. Morton. E. Tavel, Clinical phonocardiography and external pulse recording, 1978.

37. McDonald, et al, A comparison of the relative value of non invasive techniques. Am. Heart J. 88:454. 1974.

38. Manolas et al, Relationship between duration of systolic upstroke of apex cardiogram and interval indices of myocardial function in man. Am. Heart J. 91:726, 1976.

39. Mounsey. Precordial pulsation in relation to cardiac movements and sound. Brit. Heart J. 21:457, 1967.

40. Meckenzie, The study of the pulse, arterial venous, hepatic in man and the movements of the heart. 1902.

41. Margolas, C, The significance of ejection period/Tension period as a factor in the assessment of cardiac function. Dis. Chest 46:706, 1964.

42. Oreshkov, V: Indirect measurement of isovolumic contraction time on the basis of polygraphy tracing (ACG, CT & PCG) Cardiologia, 47:315, 1965.

43. O, Rourke, Arterial pulse in health and disease Am. Heart Journal 82:687, 1971.

44. Oreshkov, V: A new mechano cardiographic index in evaluation of Mitral stenosis and Apex cardiographic study Am. Heart J. 79:789, 1970.

45. Oreshkov, V: Q1 or C1 interval in the diagnosis of mitral stenosis. British Heart J. 29:778, 1967.

46. Robinson, B, The carotid pulse. II Relation of External recordings to Carotid, Aortic and Brachial pulses. British Heart J, 25:61, 1963.

47. Spodick, D.H., et al, Pulsus alternans: Physiologic study by non invasive techniques. American Heart J.; 88:766, 1970.

48. Sutton, R, et al: Correlation of pre ejection period with left ventricular ejection fraction. Clin. Res., 13:28, 1970 (Abstract).

49. Samuel Zoneraich: Non invasive methods in cardiology, 1974.

50. Shah, P.M. et al: Determinants of atrial (S4) and ventricular (S3) gallop sounds in primary myocardial disease. N. Engl. J. Med. 278:753, 1968.

51. Wallace, A.G., et al: Duration of the phases of left ventricular systole. Circ. Res., 12:611, 1963.

52. Weissler, et al: Systolic time intervals in Heart failure in man. Circulation, 37:149, 1968.

53. Weissler, et al: Beside techniques for the evaluation of ventricular function in man. American J. Cardiol, 23:577, 1969.

54. Weissler, et al: Systolic time intervals in cardiac disease. Mod. Concepts Cardiov. Dis., 40:1, 1970.

55. Weissler, et al: Relationship between left ventricular ejection time, Stroke volume and the heart rate in normal individuals and patients with cardiovascular disease. American Heart Journal 62:367, 1961.

56. Weissler, et al: Effect of digitalis on systolic time intervals in heart failure. American J. Med., 259:4, 1970.

57. Willems, et al: On the value of Apex cardiography for timing intra cardiac events. American J. Cardiol, 28:59, 1971.

58. Wiessler et al: Systolic time intervals as a measure of left ventricular performance in man. Progress in Cardiology, I 1972.

————:o:————