Non-Invasive Assesment of 100-Normal Males

By

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INTRODUCTION

In the history of medical practice, analysis of arterial pulse, as an indication of health or disease has been in practice since centuries. However it was not untill 1860, when MAREY analysed arterial pulse for specific cardiovascular disorders. GARROD, fourteen years later, conclusively reported for the first time, the relation of ventricular ejection time to the heart rate. This milestone in cardiology was followed by WEISS and JOACHIM report of 1911 on the delay in 1st heart sound. Analysis of the ventricular systole by using ECG, PCG, Arterial pulse recordings were reported by KATZ and FEIL. The effects of sex and posture on ejection time were described by LOMBARD et al: in 1926.

INVERSE relation of Q1 to cycle length in mitral stenosis with atrial fibrillation, was reported by SPRAGUE & CO in 1951, However fresh light was injected in Apex Cardiography by BENCHIMOL & DIAMOND in 1962, by using ACG, for the evaluation of Systolic time intervals. Intensive trials and study by WEISSLER in non invasive assessment of Cardiovascular function, made it possible to better understand the systolic time intervals and their application in clinical practice.

It is known that after the initial period of Electro mechanical delay (Electro pressor latent period — QC interval) the initial phase of ventricular contraction, (pre-iso volumic contraction time — CM1) starts with the contraction and tension of septal, papillary and ventricular muscles, supplemented by the tension in the chordae tendineae and the valvular cusps. This builds up pressure in the ventricles. When this pressure rise is equal to or just crosses over the pressure in the atria, mitral and tricuspid valves close. Following the closure of these valves, pressure in the ventricles continues to rise. As soon as it reaches or crosses over the pressure in great vessels the aortic and pulmonary valves open and ventricular ejection starts. The phase from the mitral and tricuspid valve closure to the opening of aortic & pulmonary valve, is known as pressure elevation time (Isovolumic contraction time—M1E).

To begin with, the ejection from ventricles is rapid and major ventricular contents pass out in the great vessels in the early phase of ejection. When the pressure in the aortia & pulmonary, vessels increases beyond the pressure in the emptying ventricles, aortic and pulmonary valves close, which prevent the backflow. The pressure continues to decrease in the

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ventricles during the early part of diastolic phase (Iso volumic relaxation time). As soon as it is equal or little less than the pressure in the atria, the mitral and tricuspid valves open. Because of existing pressure gradients across the A.V valves by this time most of the atrial blood (60-65%) rushes to the ventricles without any active atrial contraction. This is followed by the phase of slow ventricular filling and filling is completed by the atria' contraction during the late part of ventricular diastole.

Understanding of these pressure changes and their timings, will give a better insight on our paper, which purposes, to understand the phases of systole, diastole, apex cardiographic events, arterial pulse wave changes in healthly individuals of our population.

MATERIAL AND METHOD, USED.

A. Subject Selection:

100 adult males between ages 21-30 (Table-I) were selected amongst the young doctors, senior medical students, technicians, ward servants and healthy patient-attendants, representing different geographic, social & racial sections of the province. Each had thorough history and medical examination, ECG, often X-ray chest, even M-mode ECHO check to rule out any diasease (i-e MVP etc), before enlisting them into the study.

B. Equipment and Method:

MINGOGRAPH-81, ELMA SCHONDER POLYGRAPH, 8-CHANNEL machine with multiple band pass filters for heart sound recordings and facility to record, ECG, APEX

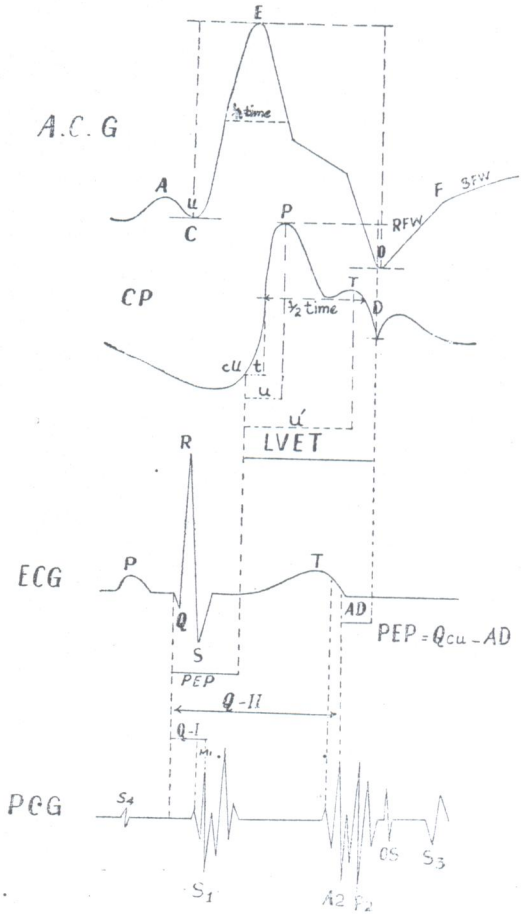

Diagram 1:

CARDIOGRAM and ARTERIAL WAVES, was used. All the recordings were done between 8-10 AM to avoid diurnal variations. The subjects were put in semi recumbent, left lateral position, supported with pillows to the back. The apex beat was located by palpation and pizo electric crystal microphone placed at the site of maximum impulse. Right Carotid pulse was palpated and button-shaped

pick up was placed over the carotid just above the right clavicle. Heart sound microphone was placed at the left mid precordium for recording the heart sounds. All the recordings were done with speed of 50-100 mm/sec in held expiration. Inadequate or defective paper recordings were taken out of the study. For good recordings, subject selection was done carefully so as to have good Apex cardiogram and Heart sound recordings.

C. Abbreviations Used & Methods of Measurments of various Cycle Lengths:

1. B.S.A. — Body surface area.
2. B.P — Blood pressure.
3. H.R. — Heart rate.
4. Q.R.S — From 'Q' of ECG complex to the end of 'S' wave.
5. QR — From 'Q' of QRS complex to the peak of R wave.
6. PR — From the beginning of 'P' wave of ECG to the beginning of Q wave.
7. RR — From the peak of 'R' of one QRS complex to the peak of R wave of next QRS.
8. ACG — Apex cardiogram.
9. QC — From 'Q' of QRS complex to C-point of ACG.
10. C-M1 — From 'C' of ACG to first major component of Ist heart sound (Mitral).
11. M1E — From the mitral component of Ist heart sound to Aortic component of Ist heart sound or 'E' of ACG.
12. QS1 — From the 'Q' of QRS complex to the mitral component of Ist sound.
13. I.V.C.T — Iso-Volumic contraction time (M1E).
14. TRUE IVCT — From "C" of ACG to the 'E' of ACG (CM1 + M1E) of QA2—LVET or QE—QC.
15. L.V.E.T — (Left ventricular ejection time) from the beginning of carotid upstroke wave to the dicrotic notch.
16. P.E.P — (Pre—ejection period)
    — QC + CM1 + M1E
    — or QE ('E' OF ACG)
    — or 'Q' to the beginning of carotid upstroke minus P.T.T (pulse Transmission Time).
    — or QS2—LVET
17. Q-A2 (Q-S2) — From the 'Q' of ACG to the aortic component of second Heart sound.
18. S1 — S2 — From the mitral component of Ist sound to the aortic component of 2nd sound.

19. IIA—0 INTERVAL — (Iso volumic relaxation time). From aortic component of 2nd sound to the 'O' point (Mitral valve opening of ACG.
20. OF — (Rapid filling time). From the 'O' point of ACG to 'F' point of ACG.
21. S.F.W — (Slow filling wave). From the 'F' point of ACG to the beginning of 'A' wave of ACG.
22. 'A' WAVE OF ACG — From the end of slow filling wave to the 'C' point of ACG.
23. P-A — From beginning of 'P' wave of ECG to the beginning of 'A' wave of ACG.
24. T-A INTERVAL: — From the end of 'T' wave of ECG to the aortic component of 2nd Heart sound.
25. A-Q INTERVAL — From the beginning of 'A' wave of ACG to 'Q' of ECG.
26. II A — S3 — From the aortic component of 2nd Heart sound to S3 of PCG.
27. II A — S4 — From the aortic component of 2nd Heart sound to S4 of PCG.
28. S4 — S1 — From S4 to the first high frequency component of S1.
29. dp/EO — ACG systolic ascending wave amplitude/EO (Lt. ventricular retraction).
30. ARTERIAL WAVE dt/dp — Down stroke amplitude/upstroke amplitude.

Table I: General Information

No. of Individuals Assesed — 100 (All Males(.

Parameter	*Min.*	*Max.*	*Mean.*	*S.D.*
Age (Years)	21	30	25	7
Body Surface Area (m2)	1.53	1.87	1.68	0.08
Mean BP (mmHg)	73	104	92	7

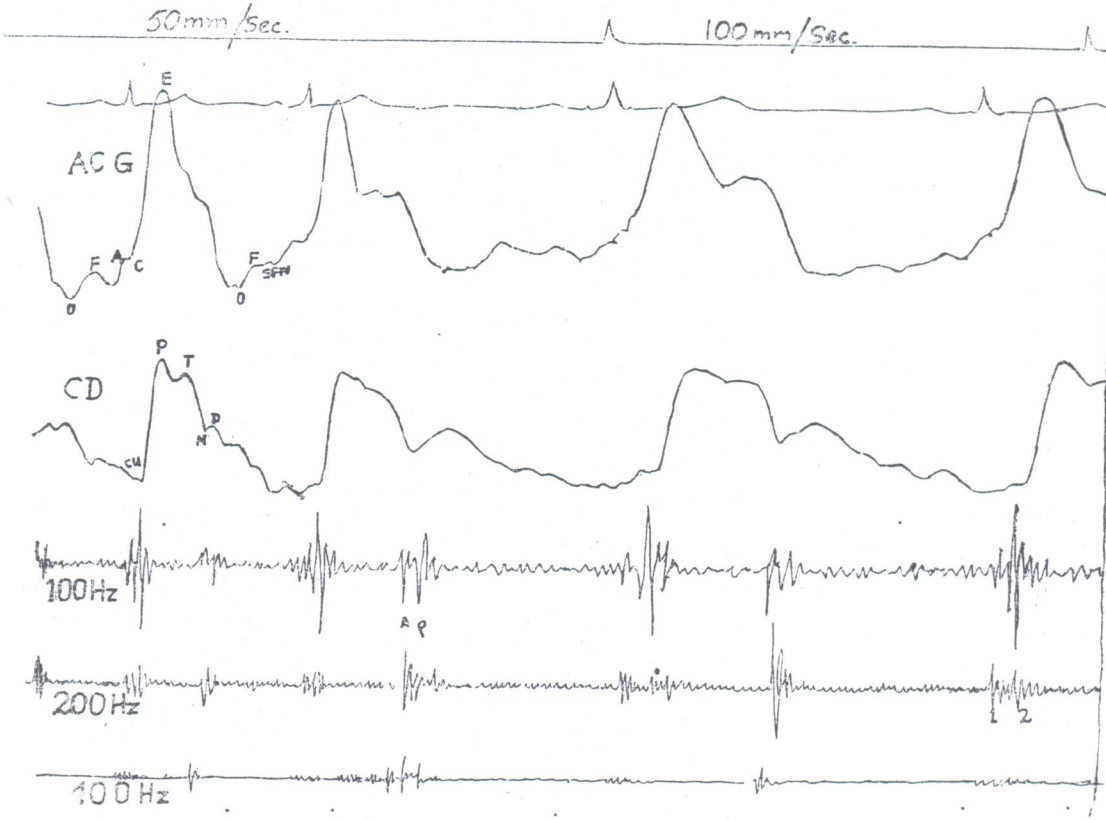

Fig. 1

Table II: Systolic Time Intervals in Normal Individuals: Regression eluation (Weissler ET, AL).

Systolic interval	*Sex*	*Regression equation*	
QS2	M	QS2 = -0.0021 HR+0.546	0.014
	F	QS2 = -0.0020 HR+0.549	0.014
PEP	M	PEP = -0.0004 HR+0.131	0.013
	F	PEP = -0.0004 HR+0.133	0.011
LVET	M	LVET = -0.0017 HR+0.413	0.010
	F	LVET = -0.0016 HR+0.418	0.010
S1S2	M	S1S2 = -0.0018 HR+0.456	0.015
	F	S1S2 = -0.0016 HR+0.461	0.012
Q-1	M	Q-1 = -0.0004 HR+0.090	0.011
	F	Q-1 = -0.0003 HR+0.089	0.009
ICT	M	ICT = 0.038	0.010
	F	ICT = 0.039	0.009

All intervals expressed in seconds.

Table III: ECG: LEAD-II.

All Values in Milli-Seconds.

Cycle Length	*Min.*	*Max.*	*Mean.*	*S.D.*
R-R	600	1000	783	107
H.R	60	100	78	10.7
QRS	60	80	75	7
Q-R	30	40	34	4
P-R	130	160	151	9

All inter pretations were done in consecutive, 5—cycles; and average values of each subject were then accepted as single value for that subject. All hundred tracings were dealt equally.

WEISSLER'S REGRESSION EQUATION (Table-II) was applied to all the major values so as to know the predicted values for the particular heart rate & sex, and for comparison of observed values.

All interpretations done on heart sounds presented here are in frequency of 200 cycles/sec.

Recording from normal subject with ECG, ACG, Carotid pulse and PCG was done as shown in figure 1. and measurement was done as shown in Diagram 1.

Table IV Systolic Time Intervals.

Cycle Length	*Min.*	*Max.*	*Mean.*	*S.D.*
Q-C	30	50	45	4
Q-C % R-R	4	7	6	0.64
C-M1	15	30	22	4
C-M1 % R-R	2	4	3	0.34
M1 - E.	35	65	48	7
M1-E % R-R	5	7	6	0.4
C-M1 + M1-E	50	85	70	10
C-M1+M1-E % R-R	8	110	9	0.6
P.E.P.	90	35	114	12.5
P.E.P % R-R	13	316	14.5	1
L.V.E.T.	230	20	274	27
L.V.E.T % R-R	30	42	35	3
P.E.P/L.V.E.T	36	48	42	3
Q-S1	55	75	66	6
Q-S1 % R-R	7	10	8	1
Q-S2	330	440	388	37
Q-S2 % R-R	44	56	50	4
S1-S2	270	370	320	33
S1-S2 % R-R	37	47	41	3

Table-V

Indices	*Min.*	*Max.*	*Mean.*	*S.D.*
C-M1/M1-E%	37	55	45	5
M1-E/L.V.E.T%	14	21	17	2
Q-S1/IIA-0%	63	94	75	7
PCG Index (Q-S —(11A-0)	—10	—35	—23	7
E. F	0.52	0.68	0.6	0.04

Table VI Diastolic Time Intervals.

Cycle Length (In Milli Seconds) With % R §R	*Min.*	*Max.*	*Mean.*	*S.D.*
S2-S1	315	640	460	86
S2-S1 % R-R	52	64	59	4
11A-0	75	100	87	8
11A-0 % R-R	9	13	11	1
0-F (RFW)	40	90	63	14
0-F % R-R	7	9	8	1
S.F.W	90	375	226	77
S.F.W % R-R	15	37.5	29	6
A-Wave (Breadth)	50	80	65	7
A-Wave % R-R	8	8	8	0
PR-OF	60	120	85	18
P-A	110	140	129	8
A-Q	20	30	23	5
T-S2 (A)	40	60	50	6

Table VII Apex Cardiographic Values.

	Min.	*Max.*	*Mean.*	*S.D.*
A/EO %	3	11	7	2
OF/EO %	3	15	11	4
B-S2/S1-B %	53	72	63	5
CE/E.O %	55	95	75	12
½ Time % R-R	17	30	25	3
(C-M1) - OF msec.	—25	—60	—40	9

Table VIII: Corotid Pulse.

Variables.	*Min.*	*Max.*	*Mean.*	*S.D.*
t Time (msec)	15	30	24	5
U Time (msec)	50	80	68	9
U1 Time (msec)	190	270	243	22
L.V.E.T - U1 (msec)	40	65	52	7
Initial Rate of Rise (mmHg/sec)	500	1333	950	244
dt/dp% Amp: Syst. Down Stroke / Amp: Syst. up Stroke	42	90	62	11
1 Time % - R-R	29	31	30	0.6
D/dp% Amp. D-Wave / Amp. Syst. up Stroke	5	13	11	5

RESULTS

For the Assessment of STIs in 100 normal males in this study with results (Table I—X) we used ECG, PCG, ACG, Rt. external carotid pulse. Our results do not differ much from the results of previous workers in this field. The minor differences noted are due to short BSA, for the subjects in study, in our populations with short LVET & prolonged PEP.

The data presented was scrutinized independently by author & co-authors and the statistical analysis performed by the statistics department, with 'p' values between 0.01 to 0.04.

Table IX. In Milli Seconds. Electro Arterial Wave Intervals

'Q' of ECG to Arterial Wave

Cycle Length.	*Min.*	*Max.*	*Means.*	*S.D.*
CD	120	160	134	9
Brachial	150	200	177	12
Radial	170	230	202	15
Finger	180	280	230	24
Femoral	160	220	190	17
Post. Tibial	200	330	291	24
Dorsalis Pedis.	270	370	314	39
Pulse Transmission Time.	10	35	24	6
Pre Ejection Period.	90	135	114	13

Table X. Peripheral Pulse Waves.

Variables.	*Mean dt/dp* %	*Mean 1 Time* % *R-R*	*Mean D/dp.*
Brachial.	63	25	10
Radial.	80	23	11
Finger.	68	26	11
Femoral.	73	29	8
Post. Tibial.	101	20	15
Dorsalis Pedis.	92	22	14

DISCUSSION

The cardiac pump performance behavior, depends on the heart rate, pre load, after load, contractile strength of myocardium and synergy. Non invasive assessment by using ECG, PCG, ACG and carotid pulse wave recordings, helps in quantifying and grading the cardio vascular function. Most of the subjects in the study inspite of reassurance were under stress, therefore increased catecholamine secretion and increased heart rate. The Body surface area of our subjects in the age group of 21-30 years is definitely lower than that of the western population.

Table XI.

MEAN PREDICTED AND OBSERVED VALUES (IN MILLI SECONDS)

For Major Cycle Lengths.

Cycle Length.		*Min.*	*Max.*	*Mean.*	*S.D.*
Q S1	Predicted	52	66	59	4
	Observed	55	75	66	6
Q S2	Predicted	336	420	382	22
	Observed	330	440	388	37
P.E.P	Predicted	91	107	100	4
	Observed	90	135	114	12
L.V.E.T	Predicted	243	311	281	19
	Observed	230	320	274	27
S1 - S2	Predicted	276	348	316	19
	Observed	270	370	322	33
P.E.P/L.V.E.T	Predicted	34	36	35	0.82
	Observed	36	48	42	

Predicted values drawn by applying WEISSLER'S REGRESSION EQUATION in relation to H.R. & Sex.

Short LVET in our subjects relates to comparative increase in heart rate, resulting in increased P.E.P which is mainly seen in 'QC' and CM1 intervals. This altered, CM1/M1E ratio and QS1 interval.

—All subjects have a PCG index of less than—10, suggestive of mobile mitral valve and comparatively reduced EF, because of increase in PEP/LVET ratio. Almost all systolic time intervals vary with the heart rate-except M1E interval, which showed ppoor correlation with the heart rate as observed by the western workers. Same was observed here.

The diastolic time intervals show wide variation in S2—S1 interval, disproportionate to the heart rate mainly involving SFW.

Inverse relation ship exists between I.V.R.T,. I.V.C.T, PEP and LVET.T-S2 (A) interval is directly related to LVET-U7.

None of our subjects had A/EO ratio more than 11 (related to LVEDP) and OF/ED ratio more than 15 (related to Lt. Atrial pressure). None of our subjects had S3, S4 recordable in frequency of 200 cycles/second, under study.

The change of slope of ACG systolic wave, occured beyond mid systole in all the subjects. The initial rate of rise of carotid pulse wave, minimum and maximum values are nearly same as in western population. The rate of downstroke/upstroke of carotid to peripheral vessels decreases, with momentum

of rise at radial, femoral and posterior tibial. The dicrotic wave becomes more prominent at peripheral sites. The femoral pulse wave occurs about 10 msec earlier than radial, and posterior tibial also occurs earlier than dorselis pedis.

CLINICAL IMPLICATIONS

Non invasive assesment of 100 males between the ages of 21-30 years with values of various phases of cardiac cycle presented here, reflect various cardiac cycle lengths in our society, in particular age group. The minor changes seen in some of the values are basically due to, short LVET, thus prolonged PEP and smaller body surface area of the subjects in study. 5 msec + errors in visual calcula-, tions should be acceptable as human error.

STIs have been used since long time for assesment of cardio-vascular function; except for a short dull period, momentum has re picked up with the development of Micro tip Catheters and Co-study with ultreasonics. The study of STIs with an association with ECHO and intra cardiac recordings, have not only modified the conception of origin of heart sounds but also, clinical utility of STIs in practice of medicine.

Every population should have their own standard values for their normal subjects in various age groups. This study aimed at it, may prove as a starting point for the workers in this field.

SUMMARY

Wide variety of techniques, both invasive and non-invasive, are used to asses the Cardiovascular functions. The invasive techniques, do cause some discomfort to the patients and to some extent some of them carry certain percentage of risk to life. The non invasive techniques have been in use for long and do not cause any discomfort to the patient and carry no risk. The objective of this study is to have cardiac cycle date available for our population.

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