LEFT VENTRICULAR PERFORMANCE IN VARIOUS HEART DISEASES WITH OR WITHOUT HEART FAILURE: —An Appraisal by Quantitative One-plane Cineangiocardiology—

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SINCE as early as 1958 and 1960 quantitative angiocardigraphic methods for dimensional analysis of ventricular performance have been developed¹⁻³; however, search for sensitive yet simple parameters to measure, not only mechanical performance but also contractile state of the myocardium of the whole heart, has been a continuing and important field in cardiovascular research.

Many parameters derived from angiocardigrams can be used to assess left ventricular (LV) pump function.^{4,5} Among these, ejection fraction (EF) is a widely-used and important parameter indicating LV systolic function.^{6,7-9} However, as other parameters employed to assess LV pump function, the EF is influenced by changes in preload, afterload, heart rate and contractility.^{5,10} Mean velocity of minor LV equator shortening and mean rate of circumferential fiber shortening (mean V_{CF}) are parameters obtained from the ejection phase of LV contraction.^{5,11} Although these two parameters are considered at present to provide the most reliable measures of LV contractility in the individual patient, these are also sensitive to preload and afterload.¹² Moreover, there still exists the difficult problem of contractility measurement in patients having coronary artery disease (CAD) and regional disorders of contraction by assessment of the mean velocity of minor LV equator shortening or mean V_{CF} and the difficulty remains to be solved.

The present study was designed primarily to compare usefulness of various parameters in assessing LV performance in groups of patients with different myocardial status, that is, 1)

normal LV, 2) LV disease without clinical history of congestive failure and, 3) LV disease and congestive failure (excluding patients with mitral stenosis and valvular regurgitation). Based upon one-plane LV cineangiocardigrams in right anterior oblique position parameters were derived and analysed.¹³ Furthermore, correlation between mean V_{CF} and EF in individual patients with various heart diseases was investigated. Assessments of LV behavior in relation to different pathological stresses or lesions such as chronic pressure or volume load, myocardial changes imposed by acquired cardiac diseases and in particular, mitral stenosis, were also made.

It is noted that, in patients with chronic heart disease, there is a considerable range of abnormalities of LV performance and assessments of at least three LV ejection properties (EF, mean V_{CF} at minor equator and wall motion abnormalities) are always necessary for full understanding of the derangements of LV function. This is especially true of patients with CAD.

MATERIALS AND METHODS

Seventy-five patients, 15 to 67 years of age, were studied during diagnostic left heart catheterization.¹⁴ Their diagnoses are listed in Table I. Group 1 consisted of 13 patients in whom no evidence of significant cardiac abnormality was found on complete right and left heart catheterization and/or on coronary cinearteriography. They were considered to have normal mechanical performance of the LV. Of these, eight patients had atypical chest pain but normal coronary arteriograms and one patient had functional

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TABLE I CLINICAL MATERIAL AND LEFT VENTRICULAR VOLUME, PRESSURE AND WORK IN 75 SUBJECTS

Case No.	Sex	Age (yr)	Reason for study	SA	LVEDV	LVESV	SI	EF	HR	CI	SEP	VCF	LVEDP	MSER	SW
<i>Group 1. Patients with normal left ventricle</i>															
1	M	53	Chest pain	1.71	83	23	60	0.72	60	3600	0.28	1.53	10	214	100
2	M	40	Chest pain	1.55	72	17	55	0.76	74	4070	0.27	1.85	2	204	90
3	M	42	Chest pain	1.82	72	20	52	0.72	76	3952	0.29	1.97	12	179	93
4	M	37	Chest pain	2.01	73	29	44	0.60	75	3300	0.25	1.25	12	176	70
5	F	25	Functional murmur	1.45	66	28	38	0.58	82	3116	0.23	1.66	7	165	50
6	F	25	Non-specific ST-T change on ECG	1.45	72	19	53	0.74	80	4240	0.29	1.78	9	183	76
7	M	56	Chest pain	1.60	56	22	34	0.61	103	3502	0.24	1.67	10	142	67
8	M	46	Chest pain	1.62	79	30	49	0.62	77	3773	0.27	1.22	12	181	73
9	F	43	Chest pain	1.44	79	23	56	0.71	72	4032	0.26	1.73	12	215	91
10	F	40	Chest pain	1.48	84	21	63	0.75	62	3906	0.30	1.30	8	210	99
11	M	49	Variant form angina	1.81	82	16	66	0.81	65	3770	0.31	1.82	12	212	100
12	M	51	Variant form angina	1.73	67	16	51	0.76	71	3621	0.27	1.81	14	189	70
13	M	18	Prolapsed mitral valve syndrome, trivial mitral regurgitation	1.45	71	20	51	0.72	75	3825	0.28	1.50	7	182	70
<i>Group 2. Patients with systemic hypertension</i>															
Mean		40.4		1.62	73.5	21.8	51.7	0.70	74.8	3746.7	0.27	1.62	9.8	188.6	80.7
±SD		11.6		0.18	7.9	4.7	9.1	0.07	10.7	316.1	0.02	0.24	3.2	21.8	15.8
S.E.		3.2		0.05	2.2	1.3	2.5	0.02	3.0	87.7	0.01	0.07	0.9	6.0	4.4
1	F	49	Variant form angina	1.64	66	18	48	0.73	76	3648	0.26	1.74	10	185	108
2	F	60	Chest pain	1.54	65	24	41	0.63	81	3321	0.26	1.52	7	158	91
3	M	50	Chest pain	1.94	84	29	55	0.65	70	3850	0.28	1.14	23	196	150
4	M	58	Chest pain	1.70	87	25	62	0.71	72	4464	0.31	1.50	20	200	126
5	M	58	Chest pain	1.74	74	28	46	0.62	82	3772	0.29	1.15	24	159	119
6	M	47	Chest pain	1.81	92	37	55	0.60	72	3960	0.26	1.37	14	212	111
7	M	46	Chest pain	1.78	84	37	47	0.56	80	3760	0.29	1.00	11	162	115
8	M	55	Chest pain, history of CHF, generalized hypokinesia	1.77	168	115	53	0.32	65	3445	0.25	0.60	3	212	126
Mean		52.9		1.74	90.0	39.1	50.9	0.60	74.8	3777.5	0.28	1.25	14.0	185.5	118.3
±SD		5.5		0.12	33.0	31.2	6.6	0.13	6.0	347.6	0.02	0.36	7.7	23.1	17.1
S.E.		1.9		0.04	11.7	11.1	2.3	0.05	2.1	122.9	0.01	0.13	2.7	8.2	6.0

Case No.	Sex	Age (yr)	Diagnosis	SA	LVEDV	LVESV	Total SI	EF	HR	Total LV output	SEP	VCF	LVEDP	Total MSER	SW
Group 3A. Patients with mitral regurgitation															
1	F	25	RHD, MR severe, TR	1.49	196	29	167	0.85	80	13360	0.28	2.03	12	596	220
2	F	21	RHD, MR mod. severe	1.29	117	27	90	0.77	75	6750	0.25	2.11	12	360	140
3	M	30	RHD, MR mild	1.64	106	36	70	0.66	76	5320	0.23	2.08	13	304	80
4	F	19	RHD, MR mild	1.35	87	31	56	0.64	120	6720	0.25	1.39	9	224	90
5	M	49	RHD, MR mild	1.80	102	30	72	0.71	113	8136	0.27	1.67	9	267	122
6	F	15	RHD, MR severe	1.50	193	46	147	0.76	94	13818	0.22	2.25	22	668	228
7	F	32	RHD, MR mod. severe	1.45	140	43	97	0.69	120	11640	0.28	1.11	9	346	139
8	M	16	RHD, MR mod. severe	1.35	129	27	102	0.79	103	10506	0.27	2.04	5	378	166
9	M	51	Ruptured chordae tendinae, MR mod. severe, CHF	1.70	120	29	91	0.76	133	12103	0.23	1.96	15	396	136
Mean		28.7		1.51	132.2	33.1	99.1	0.74	101.6	9817.0	0.25	1.85	11.8	393.2	146.8
±SD		13.4		0.17	38.5	7.0	36.2	0.07	21.5	3155.0	0.02	0.38	4.8	146.9	51.0
S.E.		4.5		0.06	12.8	2.3	12.1	0.02	7.2	1051.7	0.01	0.13	1.6	49.0	17.0
Group 3B. Patients with aortic regurgitation															
1	M	40	RHD, AR severe, CHF	2.02	205	87	118	0.58	92	10856	0.28	1.19	33	421	200
2	M	39	RHD, AR severe, MR mild, CHF	1.51	207	115	91	0.44	97	8827	0.28	0.72	31	325	189
3	M	42	RHD, AR severe	1.59	288	111	177	0.61	90	15030	0.27	1.22	17	656	320
4	M	55	RHD, AR mild, MS trivial, SSS	1.58	115	30	85	0.74	47	3995	0.27	1.68	14	315	160
5	M	23	RHD, AR mod. severe	1.60	173	81	92	0.53	100	9200	0.28	1.11	16	329	200
6	M	28	RHD, AR mod. severe, AS mild	1.72	159	62	97	0.61	80	7760	0.25	1.46	9	388	170
Mean		37.8		1.67	191.2	81.0	110.0	0.59	84.3	9278.0	0.27	1.23	20.0	405.7	206.5
±SD		11.3		0.18	58.3	31.8	34.7	0.10	19.5	3633.2	0.01	0.33	9.7	129.5	57.9
S.E.		4.6		0.08	23.8	13.0	14.2	0.04	8.0	1483.2	0.01	0.13	4.0	52.9	23.6

Case No.	Sex	Age (yr)	Diagnosis	SA	LVEDV	LVESV	SI	EF	HR	CI	SEP	VPF	LVEDP	MSER	SW
<i>Group 4. Patients with mitral stenosis</i>															
1	F	23	MS mod. severe, decreased motion in anterior wall and posterior region	1.38	55	35	20	0.36	83	1660	0.20	1.32	8	100	20
2	M	25	MS mod. severe, decreased motion in anterior wall and posterior region	1.77	81	30	51	0.63	72	3672	0.25	1.56	6	204	60
3	M	31	MS severe, decreased motion in anterior wall and posterior region	1.33	73	50	23	0.32	113	2599	0.20	0.71	8	115	30
4	M	17	MS severe, decreased motion in posterior region	1.49	71	27	44	0.62	83	3652	0.21	1.63	7	210	60
5	F	22	MS severe, decreased motion in postero-basal region, TR	1.41	52	25	27	0.25	95	2565	0.21	1.47	8	129	40
6	M	52	MS mild	1.75	76	21	55	0.72	62	3410	0.21	1.47	12	262	70
7	M	33	MS mod. severe	1.42	62	14	48	0.77	72	3456	0.23	2.17	8	209	60
8	M	23	MS severe, decreased motion in postero-basal region	1.55	98	62	36	0.37	75	2700	0.23	0.91	6	157	40
9	M	23	MS severe, decreased motion in posterior region	1.51	123	53	70	0.75	62	4340	0.25	1.77	3	280	80
10	M	17	MS mild	1.47	63	18	45	0.71	100	4500	0.22	2.19	16	205	72
11	M	54	Postop. mitral commissurotomy, MS mild, TR	1.69	114	62	52	0.46	74	3848	0.20	1.01	12	260	78
12	F	34	MS severe, decreased motion in postero-basal region	1.40	82	36	46	0.56	71	3266	0.21	1.55	10	219	66
13	F	36	MS mod. severe, decreased motion in posterobasal region	1.60	38	17	21	0.55	87	1827	0.29	1.16	7	72	33

14	M	26	MS severe, decreased motion in posterior region	1.61	78	23	55	0.71	80	4400	0.23	1.98	9	239	97
15	F	28	MS severe, decreased motion in anterior wall and posterior region	1.51	96	29	67	0.70	62	4221	0.24	1.58	7	279	109
16	F	21	MS mod. severe	1.30	79	36	43	0.54	66	2838	0.21	1.24	9	205	58
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Mean		29.1		1.51	77.6	33.6	43.9	0.56	78.6	3309.6	0.22	1.48	8.5	196.6	60.8
±SD		10.9		0.14	22.3	15.5	15.2	0.17	14.6	881.0	0.02	0.42	3.0	64.6	24.2
S.E.		2.7		0.04	5.6	3.9	3.8	0.04	3.6	220.3	0.01	0.11	0.7	16.1	6.1
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Group 5. Patients with cardiomyopathy															
1	M	42	Idiopathic, CHF generalized hypokinesia	1.63	240	192	48	0.20	62	2976	0.33	0.32	26	145	60
2	M	35	Idiopathic, CHF, generalized hypokinesia and akinesis	1.72	342	294	48	0.14	70	3360	0.31	0.30	31	155	50
3	F	46	Hyperthyroidism treated, SSS, CHF	1.70	95	51	44	0.46	52	2288	0.25	0.90	13	176	90
4	M	41	Idiopathic, CHF, apical and posterobasal hypokinesia	1.46	147	97	50	0.34	65	3250	0.30	0.64	14	167	60
5	F	46	Idiopathic hypertrophy, CHF	1.43	72	39	30	0.45	64	2112	0.28	0.95	16	118	50
6	M	41	Idiopathic, CHF, posterobasal hypokinesia	1.75	156	87	69	0.44	65	4485	0.31	0.72	21	223	111
7	M	25	Idiopathic, CHF, generalized hypokinesia	1.56	176	139	37	0.21	103	3811	0.26	0.45	20	142	45
8	M	16	Idiopathic hypertrophy	1.35	81	37	44	0.54	78	3432	0.25	1.32	22	176	69
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Mean		36.5		1.56	163.6	117.0	46.6	0.35	69.9	3214.3	0.29	0.70	20.4	162.8	66.9
±SD		10.7		0.15	91.1	89.1	10.7	0.15	15.2	771.9	0.03	0.35	6.1	31.2	22.8
S.E.		3.8		0.05	32.2	31.5	3.8	0.05	5.4	272.9	0.01	0.12	2.2	11.0	8.1

Case No.	Sex	Age (yr)	Diagnosis	SA	LVEDV	LVESV	SI	EF	HR	CI	SEP	VCF	LVEDP	MSER	SW
<i>Group 6A. Patients with coronary artery disease but no old myocardial infarction</i>															
1	M	55	LAD and LCFX lesions	1.62	56	12	44	0.79	85	3740	0.30	176	18	147	90
2	M	51	LAD lesion	1.74	73	26	47	0.65	82	3854	0.26	1.34	14	181	110
3	M	54	LAD and RCA lesions	1.81	91	33	58	0.64	67	3886	0.28	1.33	14	207	80
4	M	61	RCA lesions	1.70	80	30	50	0.63	84	4200	0.28	1.20	9	179	71
5	M	62	LCFX and RCA lesions	1.90	62	22	44	0.67	78	3432	0.27	1.52	8	163	98
6	M	48	LAD lesions	1.90	68	23	45	0.66	88	3960	0.26	1.19	20	173	77
7	M	67	LAD and RCA lesions	1.66	102	32	70	0.69	63	4410	0.29	1.44	10	241	133
Mean		56.9		1.76	76.0	25.4	51.1	0.68	78.1	3926.0	0.28	1.40	13.3	184.4	94.1
±SD		6.7		0.11	16.3	7.3	9.7	0.05	9.5	315.1	0.02	0.20	4.6	30.9	21.7
S.E.		2.5		0.04	6.1	2.8	3.7	0.02	3.6	119.1	0.01	0.08	1.7	11.7	8.2
<i>Group 6B. Patients with advanced coronary artery disease and old myocardial infarction</i>															
1	F	63	Mild hypertension, LAD, and LCFX lesions, old MI, CHF, apical hypokinesis	1.51	75	53	22	0.29	92	2024	0.21	0.48	20	105	53
2	M	60	RCA and LCA lesions (multiple), old anterior MI, CHF, generalized hypokinesis and akinesis	1.53	205	165	40	0.20	65	2600	0.20	0.31	37	200	40
3	M	55	LAD lesion, old anterior MI, CHF, apical hypokinesis	1.75	100	50	50	0.50	62	3100	0.30	0.99	35	167	60
4	M	54	LAD and RCA lesions, anterior MI, CHF, apical akinesis	1.72	95	53	42	0.44	60	2520	0.32	0.79	17	131	70
5	M	40	LAD and RCA lesions, old anterior	1.85	112	95	17	0.15	124	2108	0.19	0.23	35	89	25

6	M	29	LAD and RCA lesions, old anterior MI, CHF, hyperlipemia	1.87	95	57	38	0.40	83	3154	0.19	1.00	24	200	78
7	M	57	Hypertension, CHF, old anterior and inferior MI, generalized dyskinesia	1.56	251	198	53	0.21	62	3286	0.38	0.28	26	139	124
8	M	44	Mild hypertension, LAD lesion, old anterior MI, apical akinesis	1.70	86	49	37	0.43	84	3108	0.28	1.06	32	132	101
Mean				1.69	127.4	90.0	37.4	0.33	79.0	2737.5	0.26	0.64	28.3	145.4	68.9
±SD				0.14	64.2	59.1	12.4	0.13	21.9	494.9	0.07	0.36	7.6	40.8	37.2
S.E.				0.05	22.7	20.9	4.4	0.05	7.8	175.0	0.03	0.13	2.7	14.4	11.4

yr=year; M=male; F=female; SA=surface area (m^2); LVEDV=end-diastolic volume (ml/m^2); LVESV=end-systolic volume (ml/m^2); SI=stroke index ($ml/beat/m^2$); EF=ejection fraction; HR=heart rate (beat/min); CI=cardiac index ($ml/min/m^2$); SED=systolic ejection period (sec/beat); LVEDP=left ventricular end-diastolic pressure (mmHg); MSER=mean systolic ejection rate ($ml/sec/m^2$); VCF=rate of circumferential fiber shortening (circ/sec); SW=stroke work (g.m./beat/ m^2); CHF=congestive heart failure; RHD=rheumatic heart disease; MR=mitral regurgitation; TR=tricuspid regurgitation; AR=aortic regurgitation; MS=mitral stenosis; SSS=sick sinus syndrome; AS=aortic stenosis; mod=moderately; LAD=left anterior descending branch; LCFX=left circumflex branch; RCA=right coronary artery; LCA=left coronary artery; MI=myocardial infarction.

heart murmur. One patient with nonspecific ST-T change on electrocardiogram (ECG) and two patients having variant form angina also had normal coronary arteriograms. The remaining one patient with prolapsed mitral valve syndrome had a trivial mitral regurgitation. Group 2 composed of 8 patients with systemic hypertension (blood pressure > 160/100 mmHg) causing pressure overload of the LV with no demonstrable CAD. Group 3 consisted of 15 patients who had valvular lesions, causing only volume overload of the LV. In this group 9 patients had pure mitral regurgitation (Group 3A), three had pure aortic regurgitation, one had aortic regurgitation and trivial mitral stenosis, one had aortic regurgitation and mild mitral regurgitation, and one had aortic regurgitation and stenosis (difference in LV-aortic peak systolic pressure, 10 mmHg) (Group 3B). Group 4 was composed of 16 patients who had significant mitral stenosis of rheumatic origin. Group 5 comprised 8 patients who had LV myocardial disease. Five had idiopathic cardiomyopathy, two had idiopathic LV hypertrophy, and one had cardiomyopathy following hyperthyroidism which was treated a long time ago. All these patients had normal coronary arteriograms. Group 6 included 15 cases and consisted of 7 patients with CAD but without evidence of old myocardial infarction (Group 6A) and 8 patients with advanced CAD who had old myocardial infarction (Group 6B). Of 59 patients (excluding patients with mitral stenosis), 18 had clinical evidences for LV failure before the study. Patients in whom there was poor visualization of the LV during cineangiocardiology and who were in auricular fibrillation at the time of the study, were excluded from the present study.

LV cineangiocardiology was performed in the right anterior oblique position (30 degree) without anesthesia. The center of the LV was estimated according to the method described by Kasser and Kennedy.¹³ The distance from the image intensifier to the center of the LV was measured and a 1-cm² cross-hatched grid was filmed at the same distance from the image intensifier. The injections consisted of 30 to 45 ml 76% methylglucamine diatrizoate (Urografin) or 40 to 60 ml 50% sodium diatrizoate (Hypaque) into the LV. Cineangiocardiology were filmed at 60 frames/sec with simultaneous recording of the ECG.

The 35-mm cine film and ECG tracing were reviewed to select the best cardiac cycle. To

minimize the effects of contrast material on ventricular function, the earliest cardiac cycles providing adequate visualization of the LV chamber were analysed. Each ventriculogram was interpreted qualitatively for vigor and uniformity of contraction at all points along the ventricular inner surface, as well for ventricular chamber size and shape. Cardiac cycles that followed extrasystole were not used. The correction factor for the one-plane cine method was determined by selecting a film strip of the 1-cm² grid which had previously been taken at the measured image intensifier-LV distance. The area of the grid projecting over the opacified chamber was then outlined and determined by planimetry. The linear correction factor was then calculated and pincushion distortion, which caused greater magnification of the peripheral field of the LV, was corrected.¹³

The following parameters were calculated or determined.

1. LV Volume, ml/m², Stroke Index (SI), ml/beat/m², EF and Cardiac Index (CI), ml/min/m².

The LV volume (V) was calculated from the ellipsoidal formula:¹³

$$V = \pi/6 LD^2 (CF)^3$$

where L is the longest measured length taken as the line from the midpoint of the aortic valve plane to the LV apex, D is the short axis derived from the planimetric area (A) and the measured longest length from the formula ($D = 4A/\pi L$). The unseen minor axis was assumed to be similar to the calculated one.^{13,15} CF is the linear correction factor. Both end-diastolic volume (EDV) and end-systolic volume (ESV) were determined. The SI and EF were then calculated:

$$SI = EDV - ESV, EF = SI/EDV$$

All calculated volume results were corrected to equal the more precise biplane method with the formula given by Sandler and Dodge.¹⁶

2. Systolic Ejection Period (SEP), sec.

This was the product of the time interval between frames and number of frames exposed between end-diastole and end-ejection during each beat selected for analysis. The end-ejection was defined as the maximum inward wall excursion determined by visual inspection of the cineangiogram. A 50 msec value, accounting for the preejection period, was subtracted from the result in all patients except for those with mitral regurgitation.¹¹

3. Mean Rate of Circumferential Fiber Shortening (mean V_{CF}), circ/sec

This was calculated as the extent of shortening of the minor internal circumference (derived from the planimetered area and the measured longest length) between end-diastole and end-systole divided by the time required for shortening (SEP).¹¹ The mean velocity of shortening of the internal circumference was divided by the end-diastolic internal circumference.

4. Mean Systolic Ejection Rate (MSER), ml/sec/m²

This was the average volume of blood ejected from the LV per unit of time (sec) and derived from the formula:¹⁷

$$\text{MSER} = \text{SI} / \text{SEP}$$

Where

SI = stroke index, ml/beat/m²

SEP = systolic ejection period, sec

5. LV End-diastolic Pressure (LVEDP), mmHg

6. LV Systolic Mean Pressure (PSM), mmHg

7. LV Stroke Work (LVSW), g·m/beat/m²

This was calculated from the formula:¹⁷

$$\text{LVSW} = (1.36) (\text{PSM}) (\text{SI}) / 100$$

Where

PSM = LV systolic mean pressure, mmHg

SI = stroke index, ml/beat/m².

8. Heart Rate, beat/min

RESULTS

The data obtained from each group of the patients is presented in Table I. For each volume measurement a value corrected by the appropriate regression formula is given.¹⁶

LV Behavior in Relation to Different Pathological Stresses or Lesions

The LV behavior in relation to the different pathological stresses was assessed by dealing with SW and LVEDV in each group of patients. In 68 patients (excluding group 6A, patients with CAD but no old myocardial infarction), the SW's were plotted as function of the LVEDV's and are shown in Fig. 1. It is apparent that five separate groups of patients can be identified from the plot although some overlaps exist because the study was performed on patients with diseases of varying severity and duration. Thirteen patients with normal LV performance (Group 1) disclosed values of 73.5 ± 7.9 ml/m² for LVEDV and 80.7 ± 15.8 g·m/beat/m² for SW. The SI and CI were 51.7 ± 9.1 ml/beat/m² and 3746.7 ± 316.1 ml/min/m², respectively. A pressure load as ob-

Fig.1. Relationship of stroke work to left ventricular end-diastolic volume in normal left ventricles, 13 cases; in those subjected to a volume overload (valvular regurgitation), 15 cases; in those with myocardial disease (primary cardiomyopathy or advanced coronary artery disease), 16 cases; in those subjected to a pressure overload (systemic hypertension), 8 cases; and in those with mitral stenosis, 16 cases.

Note the close correlation between both parameters in those with valvular regurgitation ($r=0.939$, $p<0.01$). There was, however, no significant correlation between both parameters in the patients with myocardial disease ($r=0.004$, $p>0.1$).

LV=left ventricle; CAD=coronary artery disease; S=significant; NS=not significant.

Fig.2. Relationship of stroke work to left ventricular end-diastolic pressure in each group of the patients indicated in Fig.1.

The rise in stroke work in patients with valvular regurgitation was not consistent with the rise in left ventricular end-diastolic pressure ($r=0.412$, $p=0.1$).

LV=left ventricle; CAD=coronary artery disease.

Fig.3. Relationship of left ventricular end-diastolic pressure to end-diastolic volume in each group of the patients indicated in Fig.1.

There was a close correlation between both parameters in patients with valvular regurgitation ($r=0.557$, $p=0.02$). However, left ventricular end-diastolic pressure may be normal (under 12mmHg) when end-diastolic volume is more than twice normal. No significant correlation was noted between both parameters in patients with myocardial disease ($r=0.298$, $p>0.1$).

LV=left ventricle; CAD=coronary artery disease; S=significant; NS=not significant.

served in 8 patients with systemic hypertension (Group 2) tended to increase SW and the extra work was produced without chamber enlarge-

ment, except in one patient who had congestive heart failure. A volume load, such as 15 patients with valvular regurgitation (Group 3), tended to

increase in LVEDV (132.2 ± 38.5 ml/m² for mitral regurgitation and 191.2 ± 58.3 ml/m² for aortic regurgitation) as well as SW (146.8 ± 51.0 g.m./beat/m² for mitral regurgitation and 206.5 ± 57.9 g.m./beat/m² for aortic regurgitation). The rise in SW in these patients was approximately proportional to a rise in LVEDV ($r = 0.939$, $p < 0.01$). Total SI in most of the patients with valvular regurgitations was quite increased (average 99.1 ± 36.2 ml/beat/m² for mitral regurgitation and 110.0 ± 34.7 ml/beat/m² for aortic regurgitation) and the volume of regurgitant flow might be considerable. Sixteen patients with mitral stenosis (Group 4) tended to show decreased SW (60.8 ± 24.2 g.m./beat/m²) but the LVEDV (77.6 ± 22.3 ml/m²) was almost the same as those with normal LV ($t = 0.619$, $p > 0.5$). They had lower SI (43.9 ± 15.2 ml/beat/m²) and CI (3309.6 ± 881.0 ml/min/m²) than the normal group. Sixteen patients, with LV myocardial disease secondary to idiopathic cardiomyopathy or advanced CAD with old myocardial infarction (Groups 5 and 6B), showed chamber enlargement with large LVEDV (163.6 ± 91.1 and 127.4 ± 64.2 ml/m², respectively) but there was no increase in SW (66.9 ± 22.8 and 68.9 ± 37.2 g.m./beat/m², respectively) ($r = 0.004$, $p > 0.1$). Some patients in this group had low SI as well as CI. The relation between LVEDP and SW and that between LVEDV and

LVEDP in the 68 patients are shown in Figs. 2 and 3, respectively. The rise in the SW in patients with valvular regurgitations was not consistent with the rise in LVEDP ($r = 0.412$, $p = 0.1$). The filling pressure was within normal range (under 12 mmHg) in some patients in whom there were significant regurgitation and increased LVEDV. Patients with LV myocardial disease generally had raised filling pressure (20.4 ± 6.1 mmHg for patients with idiopathic cardiomyopathy and 28.3 ± 7.6 mmHg for those with advanced CAD). They showed, however, no significant correlation between LVEDP and LVEDV ($r = 0.298$, $p > 0.1$) and there were some patients who had increased LVEDP but normal LVEDV.

LV Function in LV Diseases With or Without Congestive Failure

In order to compare usefulness of various parameters in separating groups of patients with normal LV, LV disease without clinical history of congestive failure and those with LV disease and congestive failure, LV function was quantified from cineangiograms by determining EF, mean V_{CF} and MSER. LVEDP was also determined in each group of patients. Of 31 patients with either systemic hypertension causing pressure overload on the LV or LV myocardial disease (CAD or idiopathic cardiomyopathy), 14 had clinical evidence of LV failure before the

Fig. 4. Four important parameters (A: ejection fraction, B: mean rate of circumferential fiber shortening at minor equator, C: mean systolic ejection rate, and D: left ventricular end-diastolic pressure) for evaluation of left ventricular performance in groups of patients with different myocardial status, that is, (left) normal left ventricle, 13 cases; (middle) left ventricular disease without history of congestive failure, 17 cases; and (right) left ventricular disease with history of congestive failure, 14 cases. The bars present mean values; the brackets the one standard deviations. See text.

LV=left ventricle; CHF=congestive heart failure; LVD=left ventricular disease; EF=ejection fraction; VCF=rate of circumferential fiber shortening; SER=systolic ejection rate; LVEDP=left ventricular end-diastolic pressure; NS=not significant; S=significant; HS=highly significant.

study. Parameters of these 14 patients were compared with those of the remaining 17 patients and with 13 subjects having no LV disease (Fig. 4). Patients with valvular regurgitation were not included in this analysis because EF may not represent LV function as accurately as it does in patients with other heart disease.¹⁸ In addition, valvular regurgitation can also affect MSER calculation.

EF

EF in 13 normal subjects and 17 patients, with LV disease but no heart failure, averaged 0.70 ± 0.07 (range 0.58–0.81) and 0.62 ± 0.10 (range 0.43–0.79), respectively. These values differed significantly from that of 14 patients with LV myocardial failure in whom mean EF was 0.29 ± 0.12 (range 0.14–0.46) ($p < 0.001$ in both). Difference between normal subjects and those with LV disease but no heart failure was also statistically significant ($0.01 < p < 0.02$).

Mean V_{CF}

In 13 patients with normal LV the mean V_{CF} averaged 1.62 ± 0.24 circ/sec (range 1.22–1.97). The average value for 17 patients with LV disease but no heart failure was 1.26 ± 0.35 circ/sec (range 0.30–1.76). The mean V_{CF} 's in these two groups were statistically different ($0.001 < p < 0.01$). Fourteen patients with LV myocardial failure showed an average value of 0.61 ± 0.29 circ/sec (range 0.23–1.00), which differed significantly from those of the foregoing two groups of patients ($p < 0.001$ in both).

MSER

In 13 normal subjects and 17 patients with LV disease but no heart failure, the MSER averaged 188.6 ± 21.8 (range 142–215) and 180.5 ± 28.3 (range 132–241) ml/sec/m², respectively. These two values were not statistically different ($p > 0.4$). Fourteen patients with heart failure secondary to CAD or idiopathic cardiomyopathy had an average MSER of 153.2 ± 38.0 ml/sec/m² (range 89–212) and differed significantly from the foregoing two groups of patients ($0.001 < p < 0.01$ and $0.02 < p < 0.05$, respectively).

LVEDP

Seventeen patients with LV disease but no heart failure and 14 patients with LV myocardial failure had significantly high LVEDP's (17.3 ± 8.0 , range 7–35 mmHg and 21.7 ± 9.5 , range 3–35 mmHg, respectively) as compared with 13 patients with normal LV in whom the average LVEDP was 9.8 ± 3.2 mmHg (range 2–14) ($0.001 < p < 0.01$ and $p < 0.001$, respectively). However, the difference between the former two groups was not statistically significant ($p > 0.1$).

LV Performance in Mitral Stenosis

Various parameters (LVEDV, LVESV, SEP, EF, mean V_{CF} and MSER) of 16 patients with mitral stenosis were compared with those obtained from 13 patients having no LV disease (Fig. 5). Average LVEDV was not significantly different between both groups ($p > 0.5$). However, patients with mitral stenosis had significantly greater LVESV ($0.01 < p < 0.02$) and shorter

Fig. 5. Comparison of various parameters (left ventricular end-diastolic volume, end-systolic volume, systolic ejection period, ejection fraction, mean rate of circumferential fiber shortening at minor equator, and mean systolic ejection rate) of 16 patients having mitral stenosis with those of 13 normal subjects. See text.

LVEDV=left ventricular end-diastolic volume; LVESV=left ventricular end-systolic volume; V_{CF} =rate of circumferential fiber shortening; EF=ejection fraction; SEP=systolic ejection period; SER=systolic ejection rate; MS=mitral stenosis; LV=left ventricle; S=significant; NS=not significant; HS=highly significant.

Fig.6. Correlation between mean rate of circumferential fiber shortening and ejection fraction in all 75 patients studied.

Close correlation was noted in patients with valvular regurgitation, mitral stenosis, and also in those with left ventricular disease, irrespective of the presence or absence of local wall motion abnormalities. There were, however, 11 patients who showed discordant changes in both parameters. See text.

LV=left ventricle; MS=mitral stenosis; EF=ejection fraction; VCF=rate of circumferential fiber shortening.

SEP ($p < 0.001$) than the normal LV group. EF in the patients with mitral stenosis averaged 0.56 ± 0.17 (range 0.25–0.77), which was significantly lower than that of the normal LV subjects (0.70 ± 0.07 , range 0.58–0.81) ($0.001 < p < 0.01$). In fact, 6 patients with mitral stenosis showed EF of less than 0.55. Average mean V_{CF} and $MSER$ in patients with mitral stenosis were similar to those of the normal LV group ($p > 0.3$ and $p > 0.5$, respectively). However, mean V_{CF} of below 1.20 circ/sec was observed in 4 patients with mitral stenosis. Three of these 4 patients also had abnormal EF.

Cineventriculographic Pattern

In 13 subjects with no LV disease (Group 1), the cineangiograms showed contraction taking place in a coordinated manner with integrated inward movement of the ventricular wall during ejection. However, 8 patients with advanced

CAD and ECG evidences for old myocardial infarction (Group 6B), demonstrated regional disturbances in ventricular wall motion — LV asynergy. There were hypokinesis in three, akinesis in two, hypokinesis together with akinesis in two and dyskinesis in one patient. All these 8 patients had parameters compatible with impairments of both pump and muscle performances and 7 of the 8 patients had heart failure. Five of 8 patients with primary myocardial disease (Group 5) also showed abnormal ventriculographic patterns of contraction: 4, with generalized hypokinesis and one with a similar contraction pattern but in addition had a localized akinesis. Profound impairments of LV function were also noted in these 5 patients. In one patient with prolapsed mitral valve syndrome and mild mitral regurgitation, the posterior leaflet was noted to bulge into the left atrium with

ventricular systole.¹⁹ Of 16 patients with mitral stenosis (Group 4), 11 demonstrated more or less decrease in wall motion in the posterior or posterobasal region of the LV. Three of these 11 patients also had decreased motion in the anterior wall. These patients with mitral stenosis and concomitant wall motion abnormality did not always have parameters (EF and mean V_{CF}) indicative of impaired LV function.

Mean V_{CF} Versus EF in Evaluation of LV Function

Correlation between mean V_{CF} and EF was assessed in all 75 patients including 13 subjects with normal LV, 15 patients with valvular regurgitation, 16 patients with mitral stenosis, and 31 patients with LV disease (systemic hypertension, primary myocardial disease or CAD) (Fig. 6). Of the 31 patients with LV disease, 14 had local wall motion abnormalities. The correlation between both parameters was not significant in subjects with normal LV ($0.05 < p < 0.1$). However, close correlation was noted in patients with either valvular regurgitation ($p < 0.01$) or mitral stenosis ($p < 0.01$). Significant correlation between both parameters was also observed in patients with LV disease, irrespective of the presence or absence of local wall motion abnormalities ($p < 0.01$ in both). There were, however, 11 patients who showed discordant changes in both parameters. Seven patients with normal EF (≥ 0.55) had reduced mean V_{CF} (< 1.2 circ/sec) and 4 patients had decreased EF (< 0.55) but normal mean V_{CF} (≥ 1.2 circ/sec).

DISCUSSION

The in vivo measurement of LV volume in man by quantitative angiocardiology is of definite help in evaluating pump performance of the LV.^{9,18} Important myocardial mechanics in relation to LV contractility can also be quantified from volume measurement by determining mean rate of circumferential fiber shortening of the minor equator of the LV—mean V_{CF} .^{11,12} Thus, the dimensional analysis of LV performance is essential in providing objective information on dilatation, hypertrophy, LV function, and properties and motion of the LV wall.⁹ Moreover, prognostic information about a given patient with heart disease can be obtained and determination of whether medical or surgical intervention from the prognostic view point is feasible.⁹ Although, some non-invasive methods have recently been applied to the determination of LV

function, quantitative angiocardiology at present continues to serve as an important means in comparison with and also in calibration for the non-invasive techniques.

Single plane cineangiocardiology in the right anterior oblique position, a well-validated radiographic method for determining LV volume,^{13,15,20} have several advantages including convenience, smaller dose of contrast medium, smaller dose of radiation, the potential for multiple serial observation and volume measurements 60 times per second.¹³ However, since the cine LV volume regularly overestimates the biplane LV volume, all volume measurements in the present study were corrected by the appropriate regression formula to equal the more precise biplane method.¹⁶

Significant LV disease may affect its overall systolic function, localized wall motion or diastolic properties.⁵ EF is a parameter of systolic function and end-diastolic pressure is an indicator of the diastolic properties of the LV. The EF has widely been used for assessment of LV pump function⁶⁻⁹ and its reduction has been noted to correlate significantly the extent of wall motion abnormality in the presence of CAD.⁵ Nevertheless, this parameter can be influenced by changes in preload, afterload, heart rate and contractility.^{5,10} Recently, problem of increasing concern and interest to the cardiologist in the evaluation and care of patients with known or suspected heart disease is an assessment of contractility of the myocardium.^{12,21} It is now realized that contraction of the whole myocardium reflects the summed and integrated function of its individual contractile elements.²¹

Mean V_{CF} derived from quantitative angiocardiology provides at present the most reliable measure of basal contractility of the ventricle.¹² In the present study, the velocity was divided by end-diastolic circumference and the value was expressed as a magnitude per unit of circumferential fiber length, a term analogous to muscle length per second in isolated muscle.^{11,22} Thus, quantitative comparison of LV muscle mechanics among patients was rendered possible. In 13 subjects with normal LV, the mean V_{CF} averaged 1.62 ± 0.24 circ/sec (range 1.22–1.97). The reported normal value of the mean V_{CF} was 1.50 ± 0.27 circ/sec (range 1.23–2.03) in series of Karliner et al.¹¹

In the present study we compared usefulness of various parameters (EF, mean V_{CF} , MSER and LVEDP) in assessing LV performance in groups of patients with different myocardial status, that

is, normal LV, LV disease with or without history of congestive failure. It was found that both mean V_{CF} and EF were most sensitive indicators of impaired ventricular performance, providing complete separation of patients with LV disease and congestive failure from those with normal LV. The difference between any two groups of patients in these parameters was also statistically significant. On the contrary, the values of LVEDP and MSER showed considerable overlap among the various groups of patients. It has been reported that mean V_{CF} is a more sensitive indicator of impaired contractility than the EF^{11,23}. However, calculation of the mean V_{CF} in the plane of the minor LV circumference may not reflect other regional areas of impaired or normal cardiac function which may occur in patients having CAD or idiopathic cardiomyopathy with ventricular asynergy.¹¹ We calculated the mean V_{CF} using minor internal circumference derived from the planimetered area and the measured longest length. This might minimize the effect of ventricular asynergy on calculation of the mean V_{CF} . Although we observed close correlation between mean V_{CF} and EF in various groups of patients, there still existed discordant changes of these parameters in some patients. Of 62 patients with various heart diseases, 7 patients with normal EF had borderline low or abnormal values of mean V_{CF} . In these 7 patients, 4 had LV myocardial disease and two had valvular regurgitation and one had mitral stenosis. On the other hand, 4 patients with normal mean V_{CF} disclosed borderline low or abnormal EF values. These 4 patients included one patient with LV myocardial disease and three patients with mitral stenosis. Therefore, we think that there is a considerable range of abnormalities of LV performance in heart diseases and that assessments of at least three LV ejection properties (EF, mean V_{CF} and wall motion characteristics) are essential for full understanding of derangements of LV function. These assessments require detailed analysis of only two frames of a cineangiogram and can easily be performed in the usual diagnostic cardiac catheterization laboratory.

LV behavior in relation to different pathological stresses or lesions is of interest. Patients with significant mitral valvular regurgitation, as expected, had large SI and an increase in both LVEDV and LVESV. Thus, the EF in these patients generally remained essentially the same values as that obtained in those subjects with

normal LV. This was in contrast to the patients with aortic regurgitation, in whom larger LVEDV but lower EF was noted. Because of reduction in LV afterload in mitral regurgitation, EF may not represent LV function as accurately as it does with aortic regurgitation.¹⁸ A low normal EF in a patient with mitral regurgitation may indicate more marked impairment of LV myocardial function than in a patient without mitral regurgitation.¹⁸ In significant valvular regurgitation, SW in excess of twice the normal occurred in some patients and the rise in the SW was approximately proportional to the rise in LVEDV. From the result of the present study and also of the other reports,²⁴⁻²⁷ it is clear that the appropriate adaptation of the heart to the chronic volume load is carried out by increase in chamber volume. On the other hand, in a great majority of patients with primary cardiomyopathy and those with CAD a reduced SI relative to LVEDV (reduced EF) was noted. In a case, an EF as low as 0.14 was observed. These patients showed chamber enlargement (large LVEDV) but there was no increase in SW. The LVEDP tended to be higher with respect to volume and could be elevated with normal LVEDV. It appears that ventricular hypertrophy may result in a stiff chamber which resists filling and causes an elevated LVEDP. In the LV with intrinsic disease of muscle, although SW is normal, a greater wall tension is needed to do the same work because usually there is an elevated LVEDP. Eight patients with advanced CAD and wall motion abnormalities disclosed increased LV volume and pressure, together with impaired pump and muscle performances. In the presence of ventricular asynergy there is a region of apparent lack of motion with uncoordinated ventricular contraction. Patients usually manifest symptoms of LV failure when the size of the abnormally contracting segment exceeds 23% of the LV surface.⁹ Patients with primary myocardial disease can also show asynergic patterns of ventricular contraction.²⁸ This was noted in 5 of our 8 patients and they also showed marked impairment of LV function.

LV function in mitral stenosis is of note. It has been the traditional view that the LV is small in significant mitral stenosis because it is chronically underfilled.²⁹ However, our present data like those reported by others,³⁰ yielded rather opposite results on this point. Average LVEDV was similar to the normal group but LVESV was greater in the patients with mitral stenosis. As a result, average EF was significantly

lower than that of the normal subjects. In fact, of the 16 patients with mitral stenosis three had definitely abnormal LV function (abnormally low values of both EF and mean V_{CF}) and two had equivocal impairment of LV performance (borderline low values of EF and mean V_{CF}). There were two other patients who had abnormally low EF but normal mean V_{CF} . The association of impaired myocardial function with chronic rheumatic mitral stenosis has long been suspected^{31,32} and several reports dealing with this regard have appeared.^{30,33-36} Heller and Carleton³⁰ and Horwitz et al³⁶ have found abnormalities of the localized LV wall motion in mitral stenosis.

The cause of impaired LV myocardial function in mitral stenosis is not entirely clear. It has been postulated that LV function is abnormal due to localized wall motion abnormalities, which are probably due to fibrosis near or in the papillary muscle,³⁶ or in the postero-basal myocardium adjacent to the mitral valve.³⁰ Our present study also showed wall motion abnormalities in majority of the patients. However, this ventricular asynergy seemed having little, if any, effect on either EF or mean V_{CF} value or both in our series. In mitral stenosis, although the shortened systolic ejection period of the LV would make the diastolic filling period longer and by which a more adequate diastolic inflow to the LV can be maintained, yet SV as well as SW is significantly reduced. This may leave the LV chronically underloaded and it is our impression that function of the underloaded ventricle probably can express itself as if myocardial mechanics were impaired ("lazy LV"?). The abnormal LV function in mitral stenosis might not solely be due to true myocardial dysfunction.¹⁸ However, further study to clarify this point is necessary.

SUMMARY

Quantitative one-plane cineangiocardiology in right anterior oblique position for evaluation of LV performance was carried out in 62 patients with various heart diseases and in 13 subjects with normal LV. Parameters for evaluating both pump and muscle performances were derived from volume and pressure measurements. Of 31 patients with either systolic hypertension or LV myocardial diseases (coronary artery disease or idiopathic cardiomyopathy), 14 had clinical evidence of LV failure before the study. It was found that mean V_{CF} and EF were most sensitive

indicators of impaired LV performance among the various parameters. There was a close correlation between mean V_{CF} and EF, yet discordant changes of both parameters were noted in some patients. Furthermore, wall motion abnormalities were not infrequently observed in patients with coronary artery disease or primary cardiomyopathy. Therefore, assessment of at least three ejection properties (EF, mean V_{CF} and wall motion abnormalities) are considered to be essential for full understanding of derangement of LV function in heart disease. This is especially true of patients with coronary artery disease.

LV behavior in relation to different pathological stresses or lesions, such as chronic pressure or volume load, myocardial disease and mitral stenosis, was also studied and possible cause of impaired LV myocardial function in mitral stenosis was discussed.

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