M-mode and Two-dimensional Echocardiography in Chronic Chagas' Heart Disease

A Clinical and Pathologic Study

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SUMMARY **Sixty-four patients prospectively identified as having Chagas' disease were studied with M-mode echocardiography to identify characteristic functional and anatomic features of cardiac involvement. The control groups consisted of 10 normal subjects and 16 patients with nonischemic cardiomyopathy not due to Chagas' disease. Seventeen of the patients with Chagas' disease were asymptomatic and all had normal M-mode echocardiograms. Arrhythmias or congestive heart failure caused symptoms in 47 of the patients with Chagas' disease, 18 of whom had a distinct M-mode pattern characterized by left ventricular posterior wall hypokinesis and relatively preserved septal motion. Eleven of the 47 had diffuse hypokinesis indistinguishable from the nonspecific pattern of congestive cardiomyopathy. These motion patterns were quantitated by computing the ratio of the percentage of septal systolic wall thickening to the percentage of posterior wall thickening from measurements taken at the levels of the chordae and papillary muscles. This ratio (normal 0.45 ± 0.20 [±SD]) separated symptomatic patients with Chagas' disease (arrhythmia 0.83 ± 0.66, congestive heart failure 1.50 ± 1.68) from those in whom congestive cardiomyopathy was not due to Chagas' disease (0.29 ± 0.37) (p = 0.02–0.005).**

In addition, two-dimensional echocardiograms were obtained in 41 of 64 patients with Chagas' disease. These sector scans identified apical aneurysms and/or dyskinesis in 31 patients. We also found apical abnormalities in three of seven asymptomatic patients with Chagas' disease who had normal ECG and M-mode echocardiograms.

Echocardiographic findings were confirmed in 15 patients by cineangiography, in four at autopsy and in two at aneurysmectomy. We conclude that in some patients with chronic Chagas' disease, echocardiography shows a pattern indistinguishable from that of diffuse congestive cardiomyopathy. However, in the majority, echocardiography can detect a characteristic apical abnormality that often involves the posteroinferior left ventricular wall, with the interventricular septum relatively spared. In asymptomatic patients, two-dimensional echocardiography is of particular value in detecting early changes of the left ventricular apex.

CHAGAS' HEART DISEASE is a public health problem in the South American continent, where it is estimated that more than 10 million people are afflicted.[1] This study was undertaken to assess the clinical value of echocardiography as a noninvasive tool for evaluating this condition.

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Methods

Patients

The clinical data in this study were obtained from 116 subjects, prospectively identified from the wards and clinics of the Hospital Universitario de Caracas of the Universidad Central de Venezuela. Seventy-five were considered clinically to have Chagas' heart disease or serologic evidence of past or present infestation with *Trypanosoma cruzi,* and had a positive Machado-Guerreiro serum complement fixation and hemagglutinin test (MGT) performed and interpreted according to the method of Maekelt[2] at the Institute of Tropical Medicine of the Universidad Central de Venezuela. Eleven patients were eliminated due to technically poor echocardiograms. The 64 subjects who formed the study population were divided into three groups according to clinical presentation. The

first group included 17 asymptomatic subjects (ASY), 15 of whom were blood donors whose MGT (routinely performed on all donated blood) was positive. The remaining two ASY subjects were siblings of other patients. The patients were 26–61 years old (mean 39 years), and two were female. Seven had unsuspected ECG conduction abnormalities and/or occasional unifocal premature ventricular extrasystoles.

The second group consisted of 26 patients whose symptoms were directly related to arrhythmias and/or conduction abnormalities (AR), but not to congestive heart failure (CHF). These patients were 26–64 years old (mean 47 years), and 18 were female. Twenty-two had premature ventricular complexes, most of which were multifocal or in couplets; five patients had documented episodes of ventricular tachycardias from which they were resuscitated (two have subsequently died); four had atrial extrasystoles; and one had both atrial fibrillation and atrial flutter. Two subjects received pacemakers because of symptomatic complete atrioventricular block.

The third group (CHF) consisted of 21 patients who, because of symptoms and signs of CHF (most often biventricular), were judged to be in New York Heart Association (NYHA) functional class III or IV at their initial examination. Their ages ranged from 19–62 years (mean 50 years), and 17 were male. All CHF patients had premature ventricular complexes (most multifocal) and five had atrial fibrillation. Six of these subjects died during the study period, and necropsies were performed in four. These were independently interpreted by one of the authors who has had extensive experience in the pathologic evaluation of Chagas' disease. In the AR and CHF groups, seven subjects had a history consistent with systemic embolism.

Our two control groups had negative MGT and were drawn from urban areas where there is a low probability of acquiring Chagas' disease. The first control group consisted of 16 patients (10 females) with non-Chagas' congestive cardiomyopathy (CM) of unknown etiology. Two patients were postpartum and CHF was not related to alcohol abuse. Ages ranged from 12–64 years (mean 42 years), and all patients were in NYHA functional class III or IV.

The second control group consisted of 10 subjects (N) without cardiovascular abnormalities. These patients were ages 23–62 years (mean 39 years), and five were male.

In addition to physical examination conducted independently by two or more of the authors, all subjects had chest roentgenograms, 12-lead ECGs and routine laboratory examinations. The data on each subject were reviewed and classified by consensus of the authors. Fifteen patients were not included in the final analysis because they were shown to have coronary heart disease (n = 7), rheumatic valvular heart disease (n = 5), atrial septal defect (n = 2) and Ebstein's anomaly (n = 1).

Echocardiographic Methods

M-mode echocardiograms were performed in all subjects at least once using a commercially available instrument (Smith-Kline Ekoline 20-A). In addition to the standard practice of imaging the left ventricle at the chordal level (minor axis), the sweep was continued apically to the papillary muscle level. Great care was taken to image the entire thickness of the posterior wall of the left ventricle at both levels. When necessary, a variety of transducers was used to improve the definition of the endocardium of septum and of the posterior wall. Different recording speeds were used, including a slow sweep technique (10 mm/sec) to improve the qualitative and quantitative evaluation of relative wall motion and thickness. The technique of the "T" scan of the left ventricle[3] was applied to explore the lateral limits of segmental abnormalities.

Left ventricular dimensions were obtained just below the tip of the mitral valve where the chordae tendineae were distinct; at this level both the septal and posterior wall end-diastolic and end-systolic measurements were taken. Left ventricular end-diastolic dimensions (LVEDD) were obtained at the beginning of the QRS; left ventricular end-systolic dimensions (LVESD) were obtained either where the endocardium of the posterior wall was most anterior or where the septum was most posterior at end-systole. Left atrial size was measured at the maximal anteroposterior end-systolic dimension.

Left ventricular function was estimated by the percentage of fractional shortening (%FS) as LVEDD − LVESD/LVEDD. Ejection fraction (EF) was calculated according to the Teichholz formula.[4] The mitral E-point septal separation (EPSS) was also measured.[5]

The percentage of systolic thickening of the septum (%STS) and of the left ventricular posterior wall (%STPW) was measured at the level of the chordae tendineae (minor axis) and, by angling the transducer more apically, at the level of the papillary muscles where no chordal or mitral motion could be seen. The following equation was used: $\%ST = S - D/D \times 100$, where S and D are the systolic and diastolic wall thickness measurements (in mm), respectively. As an index of relative dysfunction of the septum or posterior wall, systolic thickening ratios were obtained by dividing %STS by %STPW. In our normal subjects, this ratio was approximately 0.4 because the contraction amplitude of the posterior wall exceeded that of the septum. When the posterior wall is relatively less contractile, the ratio is higher, and when the septum is less contractile, the ratio is lower. We chose this method of quantitatively comparing regional wall motion so that we could study the unique pattern of wall motion that we observed in Chagas' heart disease.

Each measurement used in this study represents the mean of at least three cardiac cycles. All measurements were performed by the same person.

Nine months after the study began, our laboratory acquired two-dimensional echocardiography capability. This technique was used to study 41 of 64 of our patients with Chagas' disease. A mechanical sector scanner of 30° or 82° (Smith-Kline Ekosector I) was used. Two-dimensional images in five standard views[6] of the heart were obtained and recorded on video tape

for later playback and analysis. The first view was a long axis from the standard precordial position. By rocking the tip of the transducer or moving it from one interspace to another, different basal, mitral valve, minor axis, and papillary muscle levels were obtained.[6] The two-chamber and four-chamber apical views were particularly useful for studying the ventricular apex.[7] To obtain these, the transducer was placed at the point where the maximal apical impulse was felt, with the patient lying in left lateral recumbency at approximately 45°. Special care was taken to detect qualitative apical systolic motion abnormalities (i.e., hypokinesis or dyskinesis) or aneurysmal dilatation as signs of the apical lesion that has been considered a characteristic feature of Chagas' heart disease.[8] The subcostal views were obtained by placing the transducer tip under the left costal margin and slowly angling toward the left shoulder until the apex was delineated.[9] We also used the long-axis view of the heart close to the apex, as suggested by Weyman et al.[10]

An apical lesion was defined as a dyskinetic area at the apex of the ventricle involving opposing walls and demarcated by an area of normal contraction. The junction between a contractile and dyskinetic area was recognized because contrasting motion patterns often produced a "hinge point" appearance. Patients were classified as having either a definite aneurysm or apical dyskinesis according to the size and the presence of a clear hinge point in two transducer positions. Care was taken to evaluate wall motion in all views. Septal, anterior, lateral, inferior and posterior wall motion were evaluated at the basal, mitral, chordal and papillary muscle levels. Right ventricular apical dysfunction or dilatation was also noted.

Paired observations in 49 of 70 M-mode tracings and in 19 of 43 two-dimensional echocardiographic studies recorded on videotape were made by two of the authors. Interobserver agreement was 94%, and the few differences were resolved by agreement.

Angiographic and Hemodynamic Methods

Coronary arteriography was performed using the Sones technique. Left ventricular angiograms were obtained in the standard right anterior oblique (RAO) and left anterior oblique (LAO) views after injection of Renografin-76. Cineangiograms were recorded on 35-mm film at 60 frames/sec. The angiographic material was analyzed by two of the authors.

Statistical Analysis

A *t* test was used to compare the mean differences between M-mode echographic variables in normal subjects and other patient groups. The fractional standard deviation and standard error of the mean of the ratio %STS/%STPW were also calculated.[11]

Results

M-mode Echocardiographic Findings

Table 1 shows the results of the statistical analysis of data from all five groups. The LVEDD, %FS, EF, EPSS and left atrial size were not significantly different from normal in the Chagas' ASY group,

TABLE 1. *M-mode Echocardiographic Findings*

Patient group	D (mm)	S (mm)	FS (%)	EF (%)	EPSS (mm)	LA (mm)
Normal controls (n = 10)	47.1	28.9	38.2	68.0	2.2	33.7
	±6.5	±3.3	±6.0	±6.5	±2.0	±5.1
Chagas'						
Asymptomatic (n = 17)	51.5	33.7	34.7	63.4	3.8	33.4
	±5.7	±4.2	±5.6	±7.8	±3.5	±5.4
p	NS	< 0.05	NS	NS	NS	NS
Arrhythmic (n = 26)	55.9	41.1	27.1	51.3	12.6	38.2
	±7.5	±8.9	±8.3	±13.0	±5.4	±7.0
p	< 0.005	< 0.001	< 0.001	< 0.001	< 0.001	< 0.05
Congestive heart failure (n = 21)	64.9	59.0	15.3	31.0	24.0	48.4
	±7.2	±7.8	±4.9	±9.4	±7.5	±7.7
p	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001
Non–Chagas' congestive cardiomyopathy (n = 16)	62.6	52.1	17.0	35.1	20.5	41.1
	±7.1	±8.2	±6.3	±12.2	±8.4	±10.3
p	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	< 0.05

Values are mean ± SD. *p* values refer to comparisons with normal controls.
Abbreviations: D and S are the left ventricular internal dimensions in diastole and in systole, respectively; FS = fractional shortening; EF = ejection fraction; EPSS = mitral E-point septal separation; LA = left atrium.

while these values were significantly abnormal in the AR and CHF groups. There was a progressive deterioration of the indexes of left ventricular functional in the AR and CHF groups compared with the ASY group. There was no significant difference in left ventricular function and size between the Chagas' CHF group and the non-Chagas' CM heart failure group.

Qualitative assessment of segmental wall thickening and motion from long-axis sweeps at slow paper speed (10 mm/sec) disclosed three different patterns: (1) normal; (2) left ventricular hypokinetic or akinetic posterior wall with relative preservation of septal motion (fig. 1), and (3) diffuse hypokinesis with or without paradoxical septal motion (fig. 2). All 17 Chagas' ASY subjects and 13 of 26 of the Chagas' AR group had normal sweeps. The pattern of hypo/akinesis of the posterior left ventricular wall was observed in seven of 26 patients in the Chagas' AR group, and in 11 of 21 patients in the Chagas' CHF

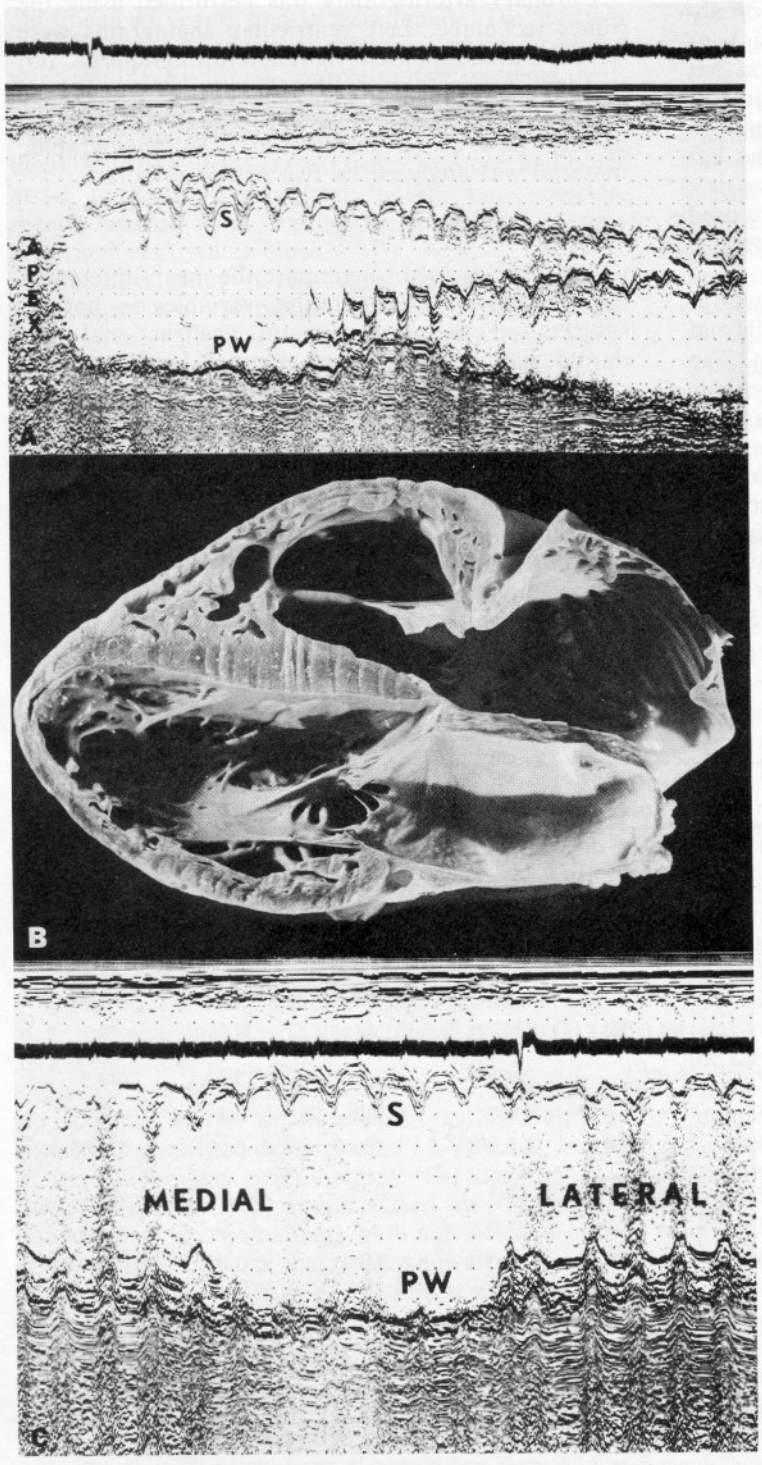


FIGURE 1. *Postmortem specimen and echocardiograms from a patient (CE) who had congestive heart failure secondary to chronic Chagas' heart disease showing the echocardiographic pattern of segmental posteroapical hypokinesis observed in nearly half of our symptomatic patients. (A) Long-axis, slow-sweep echocardiogram showing the relatively preserved systolic septal (S) motion and thickening and a thin, noncontractile posterior wall (PW). (B) The long-axis autopsy section of the heart. There is left ventricular apical and posteroapical thinning and fibrosis with relative septal sparing. The coronary arteries were normal. (C) Short-axis, M-mode "T" scan depicting the lateral extent of posterior dyskinesis and thinning. The scanning technique enhances the effectiveness of M-mode echocardiography by allowing exploration of the extent of dyskinetic areas.*

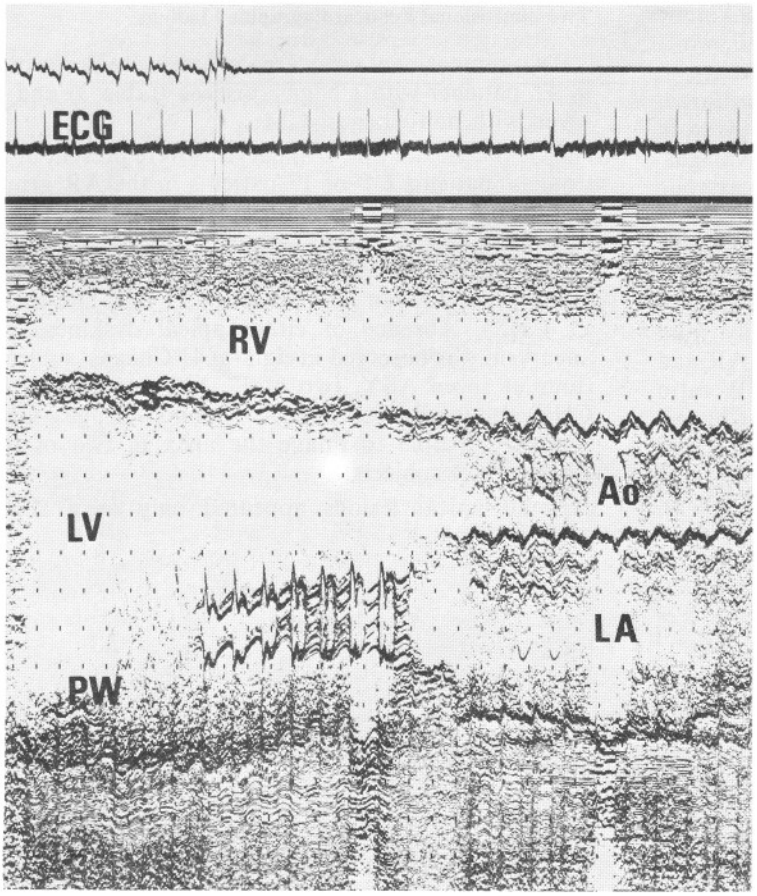


FIGURE 2. *Diffuse congestive cardiomyopathy pattern on an M-mode echocardiogram of a patient with chronic Chagas' heart disease. The diffuse hypokinesis seen here was present in approximately one-fourth of symptomatic patients. At necropsy there were diffusely thinned fibrotic walls involving all chambers and a small apical aneurysm. Amastigotes (leishmanial) forms of* Trypanosoma cruzi *parasitizing the sarcoplasm of myocardial fibers were observed in histologic sections. The coronary arteries were normal. Ao = aorta; LV = left ventricle, PW = posterior wall; RV = right ventricle; S = septum; LA = left atrium.*

group. In three of 26 patients in the Chagas' AR group and in two of 21 in the Chagas' CHF group, as well as in two of 16 non-Chagas' CM, the pattern was difficult to describe and fell between one of diffuse hypokinesis and one of a relatively hypo/akinetic posterior wall. A clearly diffuse hypokinetic pattern was observed in three of 26 patients in the Chagas' AR group, in eight of 21 in the Chagas' CHF group, and in 14 of 16 with non-Chagas' CM. To summarize, over one-third of the symptomatic Chagas' patients (those

TABLE 2. *M-mode Echocardiographic Septal/Left Ventricular Posterior Wall Systolic Thickening Ratio*

		Normal	Chagas' ASY	Chagas' AR	Chagas' CHF	Congestive cardiomyopathy
Chordal level	Mean	0.42	0.55	0.52	1.04	0.36
	SD	0.16	0.34	0.35	1.02	0.52
	SEM	0.05	0.08	0.07	0.23	0.13
	n	(10)	(17)	(26)	(21)	(16)
	p		NS*	NS*	< 0.02*	NS* < 0.02†
Papillary muscle level	Mean	0.45	0.54	0.83	1.50	0.29
	SD	0.20	0.25	0.66	1.68	0.37
	SEM	0.07	0.07	0.14	0.38	0.10
	n	(10)	(13)	(23)	(21)	(16)
	p		NS*	< 0.02*	< 0.02*	NS* < 0.005†

p values compared with normal.
†*p* value compared with congestive heart failure.
Abbreviations: ASY = asymptomatic group; AR = arrhythmia group; CHF = congestive heart failure group.

with AR and CHF) had a segmentally hypokinetic posterior wall and a nearly normal septal wall, while one-quarter showed a diffuse hypokinetic pattern.

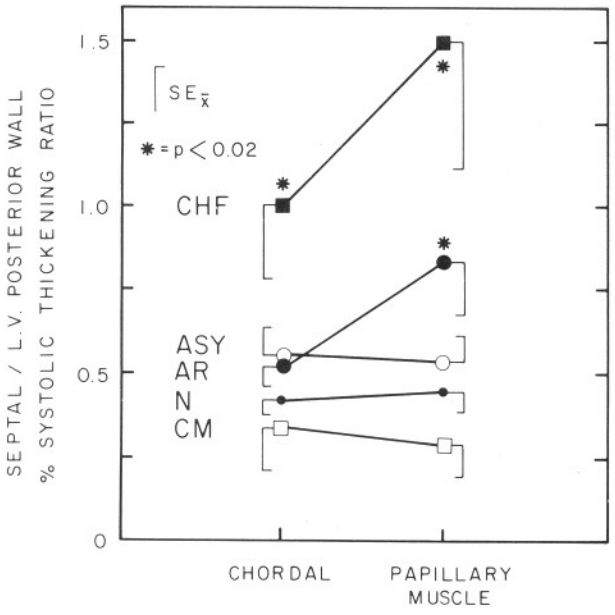
The %STS/%STPW ratios measured at the chordal and more apical papillary muscle levels are given in table 2 and figure 3. While patients with non-Chagas' CM exhibited severely depressed overall systolic thickening of both septal and left ventricular posterior walls (more pronounced in the septal than in the posterior wall), many of the Chagas' patients with AR and CHF had greatly attenuated systolic thickening of the posterior left ventricular wall with only mild depression in septal thickening. The Chagas' AR and CHF groups tended to have a %STS/%STPW ratio around 1 or higher, while this ratio in the non-Chagas' CM group was significantly different from that in the Chagas' CHF group at both chordal ($p < 0.02$) and papillary muscle levels ($p < 0.005$) (fig. 3 and table 2).

Two-dimensional Echocardiographic Findings

Two-dimensional echocardiograms were performed in 41 patients with Chagas' disease (table 3) and in seven with non-Chagas' CM.

Left ventricular apical aneurysms were present in 19 Chagas' patients (10 of 17 patients in the AR group and nine of 17 in the CHF group). Strong suspicion of dyskinetic apical motion without definitive aneurysm formation was observed in another 12 Chagas' subjects (three of seven ASY, three of 17 AR, and six of 17 CHF). Absence of either apical dyskinesis or aneurysm was observed in eight of 41 Chagas' subjects (four of seven ASY, two of 17 AR, and two of 17 CHF) and in all seven patients with non-Chagas' CM. We were unable to image the apex in two of the Chagas' AR subjects.

Small apical lesions appeared only as a slight



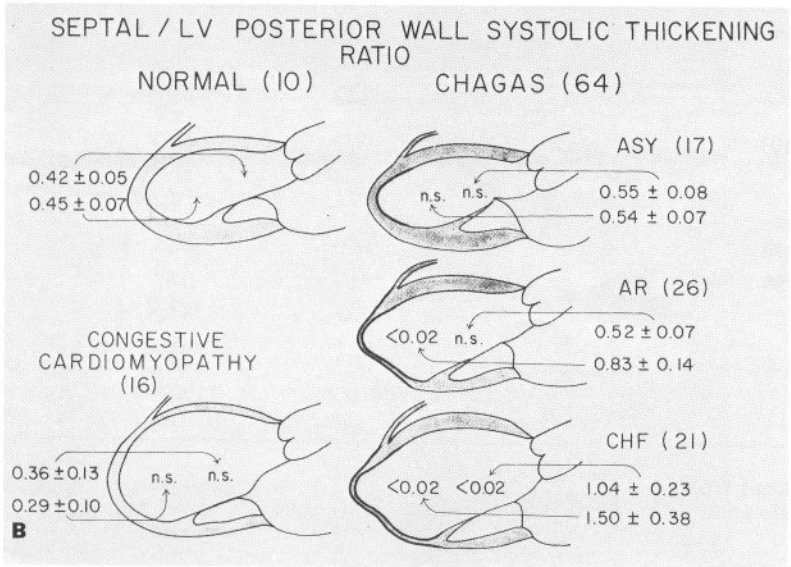


FIGURE 3. *(A) Ratio of the percentage of systolic thickening of the septum to the percentage of ventricular (LV) posterior wall thickening measured at two echocardiographic levels in patients with Chagas' disease, in control normal subjects (N) and in patients with non-Chagas' congestive cardiomyopathy (CM). Ratio values of approximately 1 (or greater) indicate a relative functional impairment of the posterior wall. These differences were observed in patients with CHF at both echocardiographic levels and in AR patients only at the papillary muscle level. (B) Graphic illustration of thickening ratios in the clinical groups. The arrows point to the region from which the ratios were calculated (mean ± SEM). CHF = congestive heart failure; ASY = asymptomatic; AR = arrhythmic.*

TABLE 3. *Two-dimensional Echocardiographic, Left Ventricular Angiographic and Coronary Arteriographic Findings in Chagas' Patients*

	Two-dimensional echocardiography			Left ventricular angiography				
Pt	Contractility pattern	Apical lesion	Other findings	Contractility pattern	Apical lesion	Other findings	EF	Coronary arteriography
				Asymptomatic				
GV	N	No	—	N	No	—	60	Normal
GH	N	D	—					
RF	N	No	—					
BP	N	No	—					
CC	N	D	—					
GM	N	No	—					
AJ	N	D	—					
				Arrhythmic				
GR	N	A	—	N	A	—	65	Normal
VC	N	NV	—	N	A	—	56	Normal
BE	N	D	—					
VB	P	A	—	PI	A	No MR	53	Normal
GM	P	D	Tr					
SA	P	NV	—	P	D	No MR	20	Normal
PV	P	A	—					
BT	N	A	—					
AJ	Di	No	—	Di	No	No MR	47	Normal
RI	P	No	pb D	pb D	No	No MR	50	Normal
PJ	P	A	Tr					
PP	N	A	—					
AJ	Di	A	Tr					
EJ	Di	A	—					
LE	Di	A	—					
LA	N	A	—	N	A	No MR	65	Normal
SM	P	D						
				Congestive heart failure				
MR	P	D	RV A					
CE	Di	No	—					
UT	P	A	RV A?	PI	Ak	MR	22	Normal
HP	P	D	RV A?	PI	D	MR	44	Normal
BZ	Di	D	—	PI	D	MR	34	Normal
PH	P	D	ps					
CE	P	A	RV A					
HJ	P	A	—	P	Ak	No MR		Normal
AJ	PI	D	Tr-RV A	PI	D	MR	43	Plaques
AC	P	A	pbD	Di	Ak	No MR		Normal
PR	P	A	RV A?					
LC	P	A						
PF	Di	A	RV A?					
PP	P	A	Tr					
GJ	Di	No						
RG	Di	D	RV A-tr					
RC	P	A	Tr	PI	A	MR	21	Normal

Abbreviations: N = normal; P and I = left ventricular posterior and inferior hypokinetic wall, respectively; Di = diffuse or generalized left ventricular hypocontractility; D = apical dyskinesis; A = apical aneurysm; Ak = angiographic apical akinesis; NV = not visualized for technical reasons; Tr = apical thrombus formation; pb D = left ventricular posterobasal dyskinesis; RV A = right ventricular apical aneurysm; ps = paradoxical septal motion; MR = angiographic mitral regurgitation; EF = angiographic ejection fraction.

dyskinetic bulging during systole. Moderate- and large-sized aneurysms, however, were easily appreciated by angiography and by two-dimensional echocardiography (figs. 4 and 5). Left ventricular apical aneurysms moved as a systolic bulging directed inferiorly, anteriorly and to the left.

Right ventricular apical aneurysms were noted in four patients and suspected in another four. All of

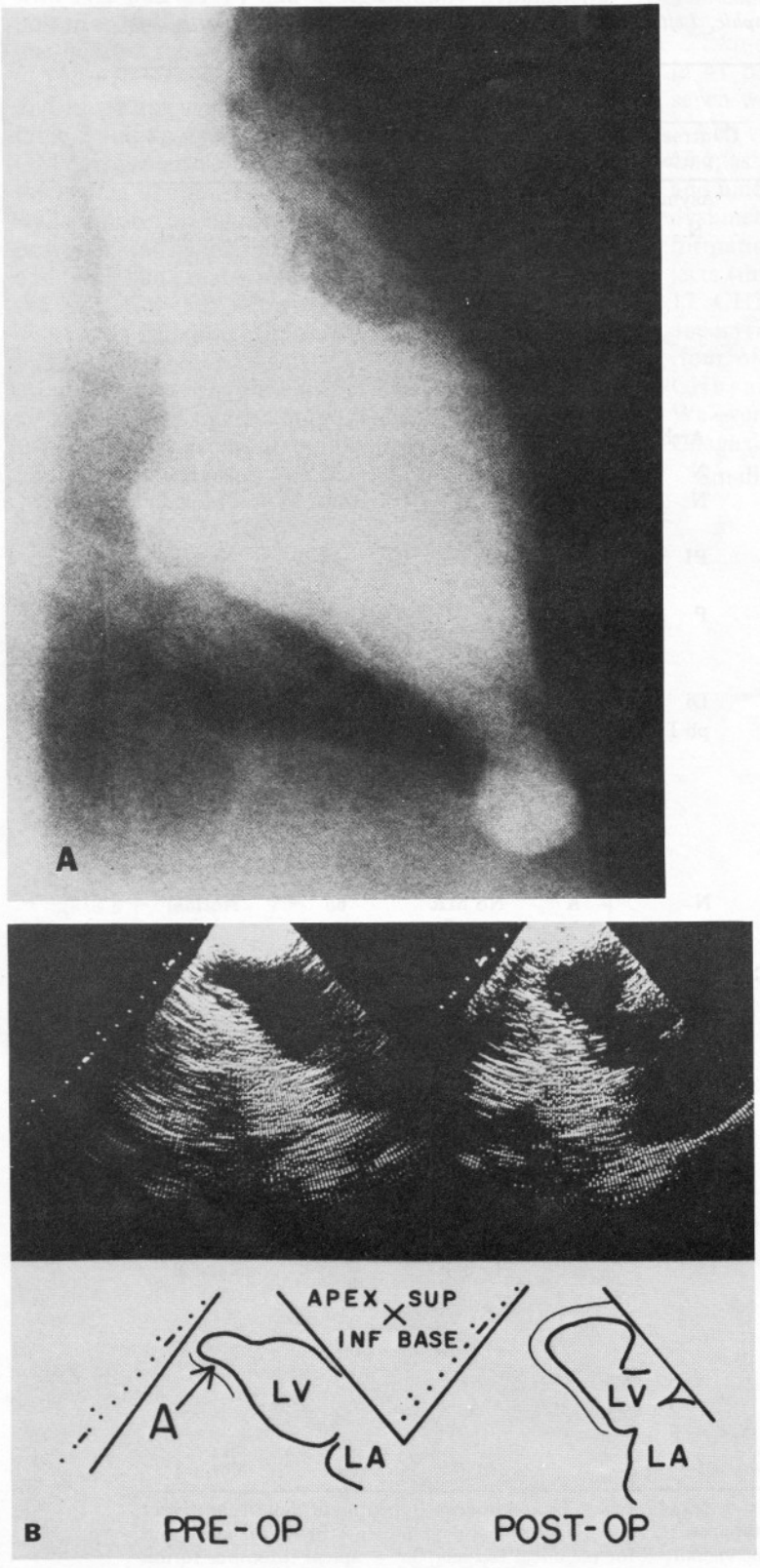
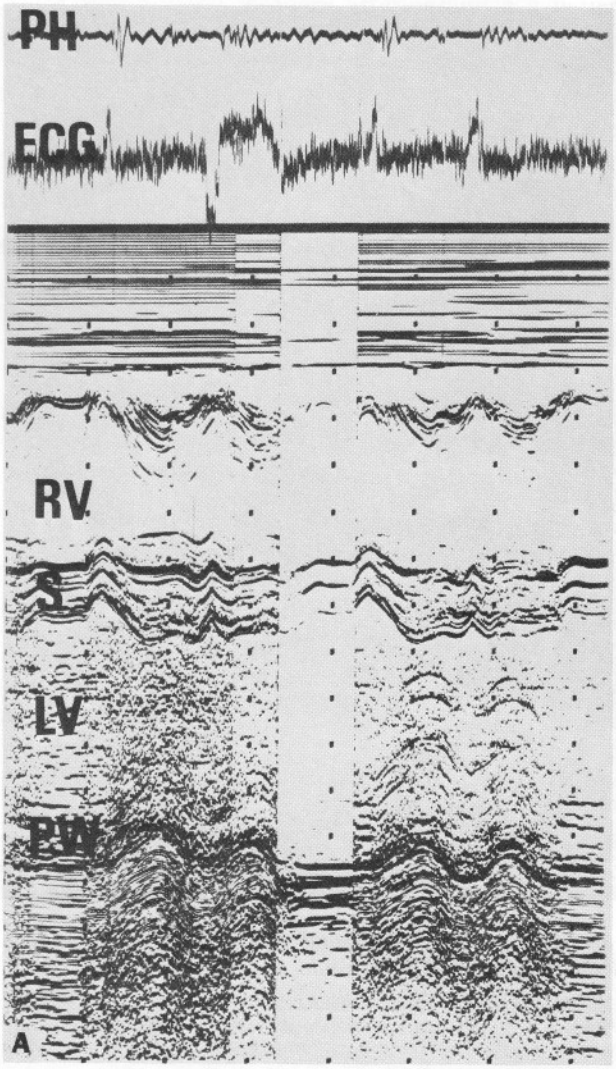
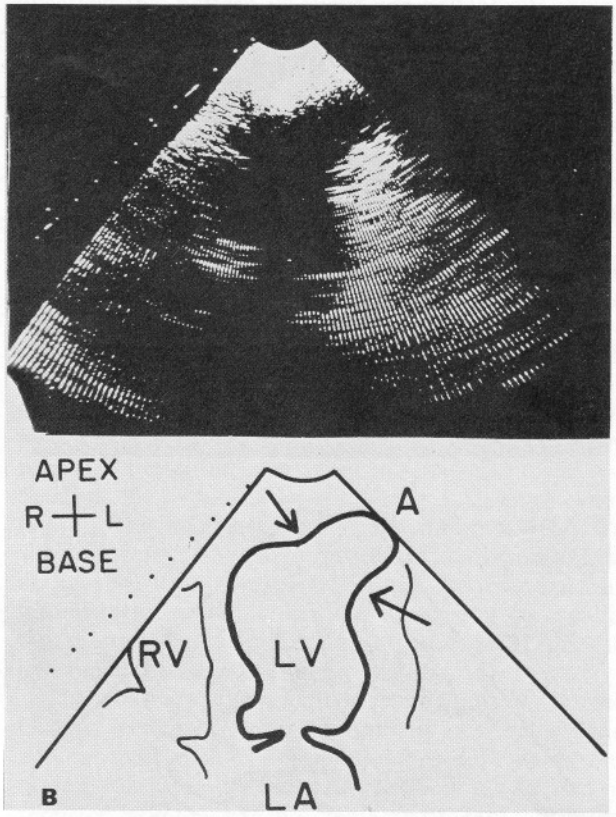


FIGURE 4. *Angiographic and two-dimensional echocardiographic studies from a patient with a moderate-size apical aneurysm whose clinical course was complicated by frequent symptomatic episodes of ventricular tachycardia. (A) The right anterior oblique angiogram showing the apical aneurysm. (B) The echocardiographic, two-dimensional, apical two-chamber view of the aneurysm (A) as seen in systole (pre-op), and the same view after aneurysmectomy (post-op). LV = left ventricle; LA = left atrium.*

these patients were in the Chagas' CHF group. Two patients had left ventricular posterobasal dyskinesis just below the posterior mitral valve leaflet, and in one it was combined with an apical aneurysm (fig. 6).

The pattern of hypokinetic or dyskinetic posterior wall motion (particularly apically) with relatively preserved septal motion was observed in 19 Chagas' patients (seven of 17 AR and 12 of 17 CHF). A diffuse hypokinetic pattern affecting all areas was present in nine Chagas' patients (four of 17 AR and five of 17 CHF) that was indistinguishable from that in seven studies performed in non-Chagas' CM. Normal wall





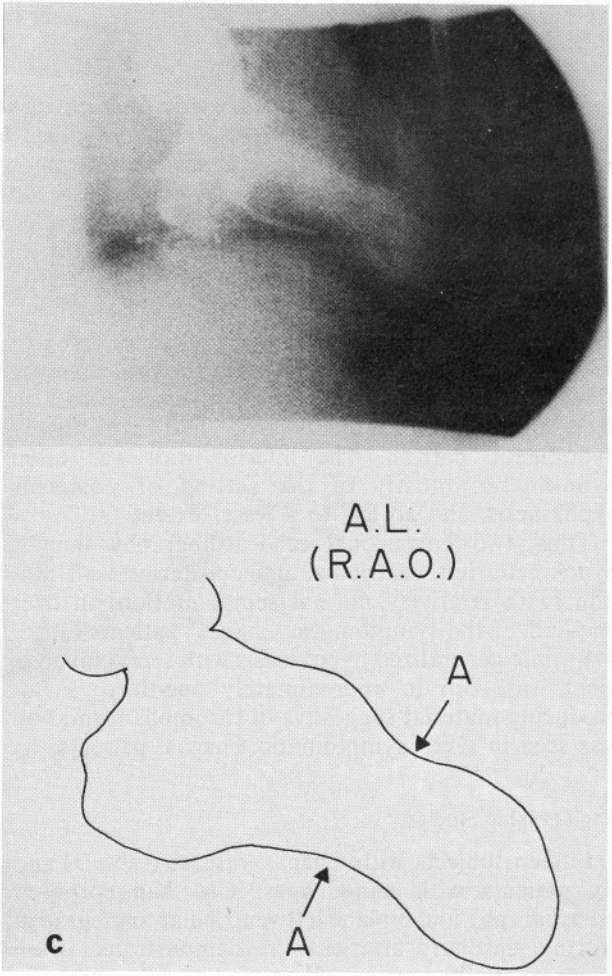


FIGURE 5. *This patient had a normal M-mode echocardiogram (A). The two-dimensional, echocardiographic four-chamber apical view (B) and the angiographic right anterior oblique view (C) showed a large apical aneurysm (A). This case illustrates the limitation of M-mode in detecting apical lesions. The only clue to the presence of the aneurysm on the M-mode was abnormally increased mitral-septal separation. PH = phonocardiogram; RV = right ventricle; S = septum; LV = left ventricle; PW = posterior wall; LA = left atrium.*

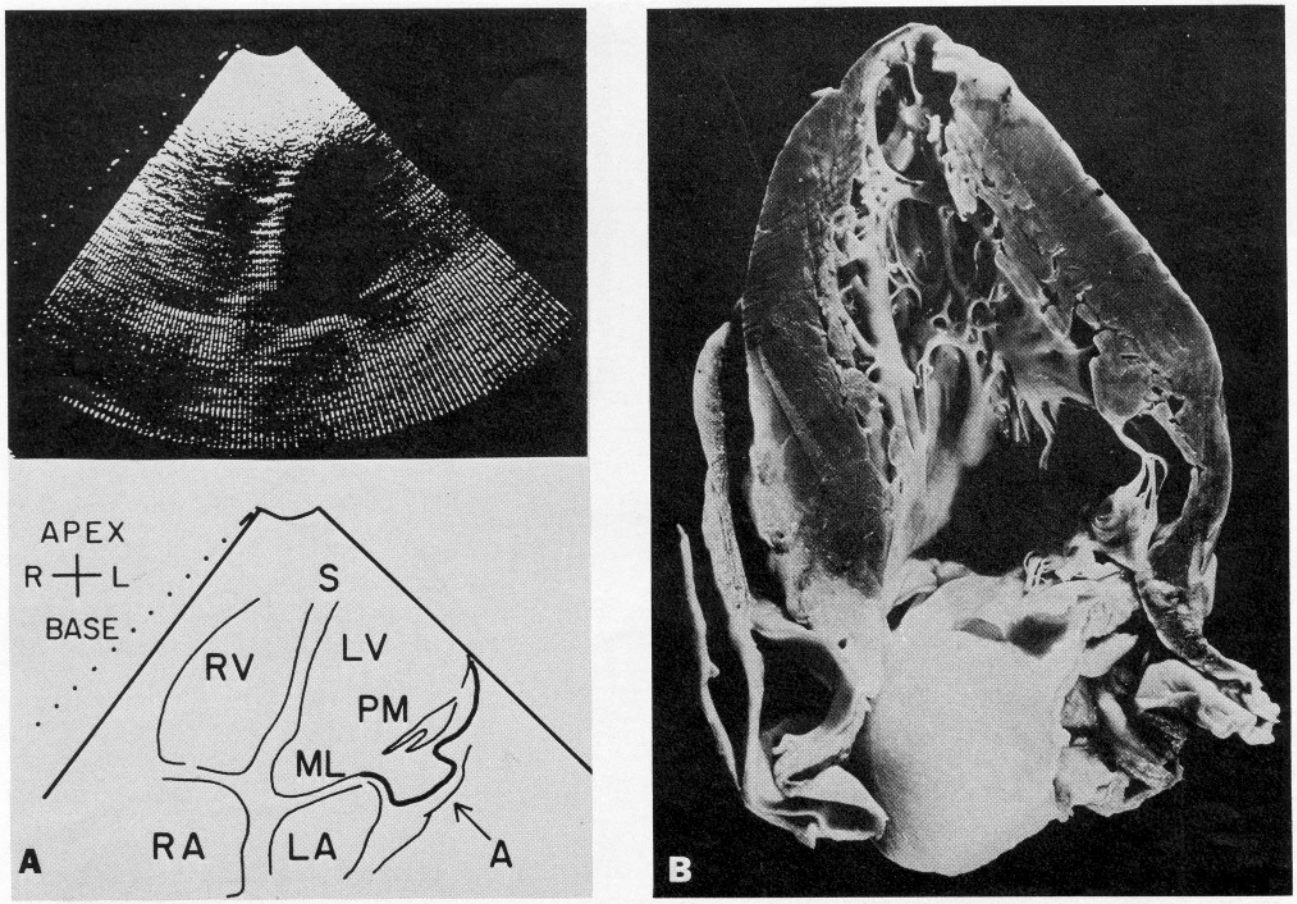


FIGURE 6. *(A) Apical four-chamber view from a patient who had posterobasal aneurysmal dyskinesis (arrow) and was clinically in the Chagas' congestive heart failure group. In real time, apical dyskinesis could also be appreciated. (B) A pathologic specimen from another patient (not in our series) shows the same combination of lesions. A = aneurysm; ML = mitral leaflets; PM = papillary muscles; RV = right ventricle; LV = left ventricle; S = septum; RA = right atrium; LA = left atrium.*

motion was present in 13 Chagas' subjects (seven of seven ASY and six of 17 AR). On the two-chamber apical view, anterolateral wall involvement was also seen in Chagas' subjects who had a diffusely hypokinetic pattern. The inferior wall was often hypokinetic, mostly in the setting of posterior hypokinesis, and always to a lesser extent.

Thus, two-dimensional echocardiography showed hypokinetic left ventricular apicoposterior wall motion (with relatively normal septal motion) in over one-half of the symptomatic Chagas' patients (19 of 34), while generalized hypokinesis with septal involvement was seen in approximately one-third. Echo-producing material suggestive of thrombus formation was seen in seven symptomatic Chagas' patients.

Angiographic Studies

Fifteen subjects with Chagas' disease (table 3) and six patients with non-Chagas' CM had coronary arteriography and biplane left ventricular angiograms. Normal coronary arteries were demonstrated in all subjects except one (an AR Chagas' patient with less than 50% obstruction of the left anterior descending coronary artery.) The RAO view was particularly useful in demonstrating the apical dyskinesis or aneurysm[13-15] present in five of seven patients in the AR group and in all seven patients in the Chagas' CHF group. None of the patients with non-Chagas' CM had apical aneurysm or dyskinesis.

Wall motion was evaluated using both the RAO and LAO views. A diffuse hypokinetic generalized motion pattern was present in all six non-Chagas' CM and in two of 15 Chagas' subjects. A hypo/akinetic posterior wall was visible in the LAO view in three of seven AR and in six of seven Chagas' CHF patients, although two had mildly hypokinetic septa. The findings from biplane left ventricular angiography and two-dimensional echocardiography are compared in table 3. There was agreement between the two methods in the location of dyskinetic areas. A source of minor disagreement between both methods was the definition of an aneurysm as opposed to apical dyskinesis.

A postmortem coronary arteriogram in a patient who had a large apical aneurysm and posteroapical wall thinning revealed normal coronary arteries with no areas of narrowing greater than 30%.

Postmortem and Surgical Correlation

In the four Chagas' CHF patients who came to autopsy and in the two Chagas' AR patients who underwent aneurysmectomy, echocardiographic location of hypo/dyskinesis correlated with the observed cardiac anatomy.

Discussion

Our results show that M-mode and two-dimensional echocardiography accurately define the anatomic abnormalities associated with chronic Chagas' disease and suggest that this form of heart disease presents a characteristic echocardiographic appearance. Apical aneurysm (with or without dyskinesis of the contiguous apicoposterior left ventricular wall) and relative sparing of the interventricular septum are typical features. This pathologic distribution of lesions is illustrated in figure 1, a postmortem specimen from one of our patients with chronic Chagas' heart disease who died from arrhythmia. Aneurysmal apical thinning and fibrosis extend through the posterior wall up to the papillary muscles and the interventricular septum is minimally involved. Therefore, it is not surprising that septal motion is relatively preserved when compared with the posterior wall, particularly toward the apex. We observed this pattern in about one-half of our symptomatic patients, while approximately one-quarter showed diffuse hypokinesis[16] and ventricular dilatation indistinguishable from that observed in non-Chagas' CM. We used drugs to reduce arterial impedance in a number of patients with diffuse hypokinesis and signs and symptoms of advanced congestive heart failure. The echocardiograms performed before and after drug therapy in three of five subjects showed improvement in septal, but not in posterior, wall motion (fig. 7). Thus, in this group the echocardiographic pattern was latent, emerging in response to vasodilation.

Relative preservation of septal motion with posterior wall hypokinesis has been observed in non-Chagas' CM when mitral incompetence is pronounced.[17] Although this situation may superficially resemble the pattern observed in Chagas' subjects, it could be distinguished by comparing systolic thickening ratios, which were significantly different (table 2). Though left ventricular posterior wall motion is decreased with preserved septal motion in inferoposterior wall myocardial infarction, this condition can usually be differentiated from Chagas' disease by clinical and epidemiologic history.

The finding of preserved septal motion in the Chagas' echocardiograms prompted us to review the angiographic studies from our laboratory and from others. Because only the RAO projection was used in reported studi3s,[13, 14] no analysis of septal motion could be made. We routinely used biplane angiography and were able to visualize areas in which the Chagas' pattern could be seen. In these cases,

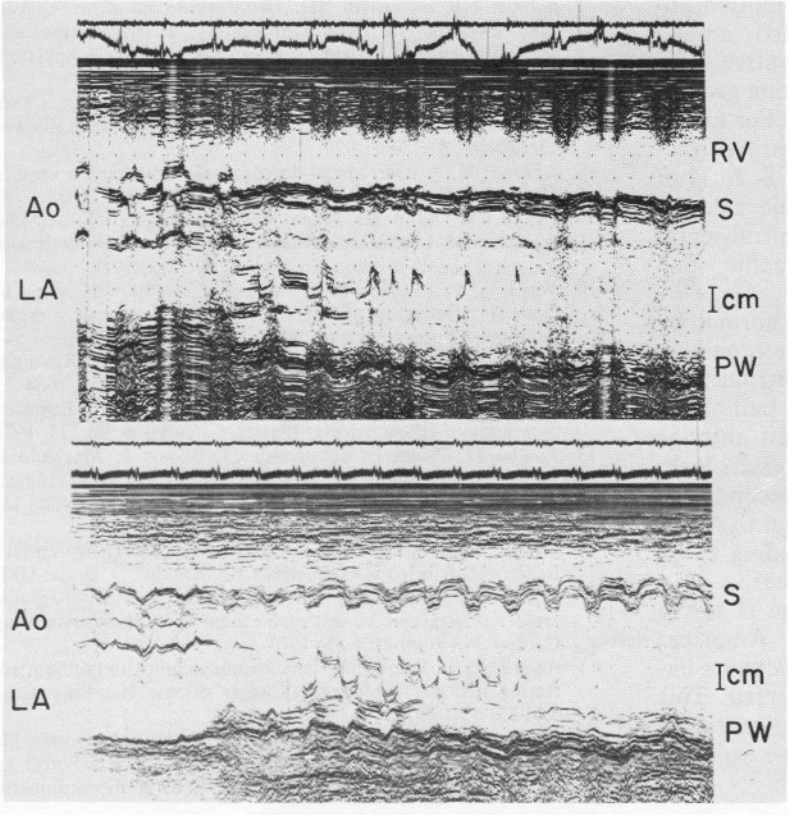


FIGURE 7. *M-mode echocardiographic slow sweeps showing the change in septal systolic motion observed after administration of oral phentolamine (lower panel) in a patient from the Chagas' congestive heart failure group. The left ventricular posterior wall remains hypokinetic despite improved septal motion. Ao = aorta; LA = left atrium; RV = right ventricle; S = septum; PW = posterior wall.*

angiographic and echocardiographic wall motion patterns correlated strongly (table 3).

An anatomic basis for our findings comes from a pathologic study of 118 autopsies of patients who died from chronic Chagas' heart disease.[19] Gross segmental, fibrotic and parietal scarring of the left ventricular apex was found in 92%, of the posterior wall in 21%, of the lateral wall in 12%, of the anterior wall in 6%, and of the right ventricular apex in 14%. These changes were not seen in the septum. However, histologic evidence of inflammatory disease (without confluent scarring) was present throughout these hearts, including the septa. This latter finding emphasized that the septum, though less affected than other areas, is nonetheless abnormal.

M-mode echocardiography appears to be of value in detecting the septal posterior wall pattern, but its major shortcoming is that it is unable to visualize the apex. Because two-dimensional studies allow apical imaging, they should be routinely used in Chagas' disease, which often will present with isolated apical lesions. As an example, one patient underwent successful apical aneurismectomy (fig. 4) because of recurrent ventricular tachyarrhythmias. The aneurysm had been initially detected by two-dimensional echocardiography though the M-mode had been normal. As three of seven asymptomatic Chagas' subjects had apical dyskinesis with normal ECGs, two-dimensional studies might be of value for long-term follow-up.

The reliability of M-mode contractility indexes is compromised by the presence of segmental disease.[4] To overcome this limitation we measured mitral EPSS, which has been shown to be a functional index relatively independent of segmental wall motion abnormalities.[5] Mitral EPSS discriminated among groups. Fractional shortening and its derivative, ejection fraction, were also effective in separating groups, but could be misleading in selected cases. For example, eight patients in the AR group had normal fractional shortening but abnormal mitral EPSS. In 11 of 13 patients with single-plane angiographic calculation of ejection fraction, the degree of mitral-septal separation correctly predicted the angiographic value (table 3).

In this series, coronary arteries were normal by angiography and necropsy. However, chest pain in chronic Chagas' heart disease may be misattributed to coronary heart disease. Because more than half of our Chagas' patients had ECG changes of left anterior hemiblock, with or without right bundle branch block, and Q waves lasting more than 0.04 second, the echocardiographic appearance of segmental dysfunction may reinforce the ECG findings, leading to an erroneous diagnosis of ischemic heart disease.

The diagnosis of Chagas' heart disease is rarely made outside of South and Central America. However, in our blood bank, 5% of blood donors living in urban non-endemic areas are infected. This suggests that a small percentage of South Americans living outside their country of origin can be expected to present with this condition.

Physical examination and electrocardiography are established screening tools in Chagas' heart disease.[20] As unsuspected advanced anatomic abnormalities are often present in the preclinical or early symptomatic periods and can be inferred from or demonstrated by ultrasound imaging, we suggest that echocardiography also be routinely used in this condition. Further, our work supports the concept that Chagas' heart disease is a progressive condition, which, though initially focused at the apex extends basally and leads to a diffuse congestive cardiomyopathy.

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