
Relationship of electrocardiographic abnormalities and seropositivity to *Trypanosoma cruzi* within a rural community in Northeast Brazil

The relationship of infection with *Trypanosoma cruzi* to ECG abnormalities was studied in a defined population in rural Bahia, Brazil. Of 644 individuals 10 years of age or older who had complement fixation tests for antibodies to *T. cruzi* and ECGs, 53.7% were seropositive. ECG abnormalities were more common in seropositive individuals than in seronegative individuals, and more common in men than in women. The peak prevalence rate of abnormal ECGs occurred among seropositive individuals between 25 and 44 years of age; in this age group ECG abnormalities occurred 9.6 times more frequently among seropositive individuals than among seronegative individuals. The most common abnormalities were ventricular conduction defects, and right bundle branch block with or without fascicular block occurred in 10.7% of the infected population. PR intervals were longer in seropositive individuals than in seronegative individuals. Ventricular extrasystoles were slightly more common in seropositive individuals. A declining prevalence rate of abnormal ECGs among older seropositive individuals suggested selective mortality due to Chagas' heart disease. (AM HEART J 105:287, 1983.)

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It is estimated that 10 million or more Central and South Americans are infected with *Trypanosoma cruzi*, the protozoan responsible for Chagas' disease.¹ Chronic cardiomyopathy is a frequent consequence of infection and a leading cause of disability and mortality in endemic areas. The clinical and ECG manifestations of all phases of Chagas' disease have been described in hospital-based and epidemiologic studies,²⁻¹³ and histopathologic correlations with ECG abnormalities have been clearly demonstrated.¹⁴ Nevertheless, there are many gaps in the current understanding of Chagas' heart disease,

especially with regard to its natural history and prognosis. To gain further insight into the evolution of Chagas' cardiomyopathy, a longitudinal study was begun in a rural community in northeast Brazil. This paper reports the results of the initial cross-sectional study. The objective was to correlate the prevalence of ECG abnormalities with seroreactivity to *T. cruzi* in a defined population using standardized epidemiologic methods.

The study area is located in the county (*município*) of Castro Alves, State of Bahia, a tropical agricultural zone in northeast Brazil located 187 km west of Salvador. The area was selected because a survey for the triatomine vector had demonstrated infestation by *Panstrongylus megistus* of 20% of the houses in the county. The study population consisted of all residents of 10 contiguous *fazendas* (farm-estates) in a 25 km² rural area. At the time of the study, the population of the area was 1051, of whom 709 individuals were 10 years or older (313 men and 396 women). The ages used in this paper are those recorded at the time of the original census of the area which preceded the clinical examinations by 3 to 5 months. Further information concerning

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Table I. Age and sex distribution of the study population and rate of seroreactivity to *T. cruzi**

Age (yr)	Men			Women		
	No. ECG done	No. seropositive	% seropositive	No. ECG done	No. seropositive	% seropositive
10-14	56	20	35.7	69	25	36.2
15-19	30	15	50.0	54	34	63.0
20-24	31	20	64.5	43	25	58.1
25-34	43	29	67.4	60	35	58.5
35-44	46	27	58.7	46	32	71.1
45-54	41	28	68.3	31	17	54.8
55-64	17	8	47.1	20	2	28.0
65-74	19	9	47.4	18	11	61.1
75+	6	1	16.7	8	3	37.5
TOTAL	289	157	54.3	355	189	53.2

*Complement fixation positive = titer of 1:8 or greater.

the population and demographic methods has been published.¹⁵

METHODS

A team of four physicians and six assistants examined one half of the study population in December, 1973, and the remainder in February, 1974. Ten cubic centimeters of venous blood and an ECG were obtained on persons 10 years of age or older. ECGs were not performed on individuals less than 10 years of age because we and others had observed a low prevalence of ECG abnormalities in young children with Chagas' disease.⁷ The ECGs were performed with a single-channel Hewlett Packard 1504A battery-operated electrocardiograph at a paper speed of 25 mm/sec. A five lead ECG (leads I, II, aV₁, V₁, and V₅) and a 30-second V₁ rhythm strip were recorded. The suitability of the abbreviated lead system and its sensitivity in diagnosing ECG abnormalities in population-based surveys of Chagas' disease have been discussed elsewhere.¹⁷

Analysis of ECGs. An amplification of the Minnesota Code¹⁸ was used to classify the ECGs. The amplified code¹⁷ includes criteria for complex rhythm and conduction disturbances adapted from the New York Heart Association¹⁹ and separate criteria for young patients. The ECGs were coded independently by two cardiologists. Discrepant readings were reviewed and a final coding was decided upon. The coding of a random 10% sample of ECGs was completely concordant when reviewed by a third physician experienced in the use of the code. Once coded, the ECGs were classified as normal, borderline, and abnormal according to the following scheme.

Normal. Absence of any alteration listed below under "borderline" or "abnormal," with normal sinus rhythm, sinus tachycardia, sinus bradycardia, or sinus arrhythmia. The following were allowable: rr' in V₁, if other criteria for incomplete or complete right bundle branch block were not met; in individuals under 35 years of age, tall R waves in V₅ (items 3-1 or 3-3 in Minnesota Code) if not associ-

ated with T wave or ST segment alterations; right axis deviation up to +100 degrees in individuals less than 15 years of age.

Borderline. Absence of any alteration listed below under "abnormal" plus one or more of the following: small Q waves (item 1-3 of Minnesota Code); indeterminate axis, tall precordial R waves without T or ST alterations in persons over 35, short PR interval, nonspecific ST segment or T wave abnormalities, nonrepetitive supraventricular extrasystoles, unifocal nonrepetitive ventricular extrasystoles, low QRS voltage, and wide P waves.

Abnormal. One or more of the following alterations: large Q or QS waves (items 1-1 and 1-2), pattern of ventricular hypertrophy (tall precordial R waves) with ST segment and T wave alterations, ST and T alterations of ischemic type, atrioventricular block, ventricular conduction defects; arrhythmias (supraventricular or ventricular tachycardia, multifocal or repetitive ventricular extrasystoles, junctional rhythm, atrial fibrillation or flutter).

Complement fixation tests. ECGs and complement fixation tests were obtained on 644 (90.8%) of the population of 709 persons aged 10 years or older. Of 65 persons not included, 41 had neither ECG nor serology performed because of refusal, absence from the area, or illness, 17 had no serological results, and seven had anticomplementary sera. The age-sex distribution of the study population is presented in Table I. No individual was diagnosed on clinical grounds as having acute Chagas' disease, and symptomatic heart disease was rarely apparent in the population.

RESULTS

Abnormal tracings. An abnormal ECG was recorded more frequently in men than in women,* in seropositive individuals than in seronegative individuals,† and in seropositive men than in seroposi-

*Chi-square test: $\chi^2 = 3.7$, $df = 1$, $p < 0.05$.

†Chi-square test: $\chi^2 = 22.5$, $df = 1$, $p < 0.001$.

Table II. Prevalence of electrocardiographic alterations according to sex and seroreactivity to *T. cruzi*

Finding	Men			Women			Total		
	Sero-negative (n = 132*)	Sero-positive (n = 157)	Total (n = 289)	Sero-negative (n = 166)	Sero-positive (n = 189)	Total (n = 355)	Sero-negative (n = 298)	Sero-positive (n = 346)	Total (n = 644)
Normal ECG	101 (76.5)†	90 (57.3)	191 (66.1)	131 (78.9)	128 (67.7)	259 (73.0)	232 (77.9)	218 (63.0)	450 (69.9)
Borderline ECG	23 (17.4)	26 (16.6)	49 (17.0)	25 (15.1)	32 (16.9)	57 (16.1)	48 (16.1)	58 (16.8)	106 (16.5)
Abnormal ECG	8 (6.1)	41 (26.1)	49 (17.0)	10 (6.0)	29 (15.3)	39 (11.0)	18 (6.0)	70 (20.2)	88 (13.7)
<i>Ventricular conduction defects</i>									
Right bundle branch block (RBBB)	0 (0)	12 (7.6)	12 (4.2)	0 (0)	8 (4.2)	8 (2.3)	0 (0)	20 (5.8)	20 (3.1)
RBBB with anterior fascicular block (AFB)	1 (0.8)	9 (5.7)	10 (3.5)	0 (0)	7 (3.7)	7 (2.0)	1 (0.3)	16 (4.6)	17 (2.6)
RBBB with posterior fascicular block (PFB)	0 (0.0)	1 (0.6)	1 (0.3)	0 (0)	0 (0)	0 (0)	0 (0)	1 (0.3)	1 (0.2)
Total RBBB	1 (0.8)	22 (14.0)	23 (8.0)	0 (0)	15 (7.9)	15 (4.2)	1 (0.3)	37 (10.7)	38 (5.9)
Incomplete RBBB	1 (0.8)	6 (3.8)	7 (2.4)	1 (0.6)	3 (1.6)	4 (1.1)	2 (0.7)	9 (2.6)	11 (1.7)
IRBBB with AFB or PFB	0 (0)	3 (1.9)	3 (1.0)	0 (0)	1 (0.5)	1 (0.3)	0 (0)	4 (1.2)	4 (0.6)
AFB	3 (2.3)	6 (3.8)	9 (3.1)	3 (1.8)	4 (2.1)	7 (2.0)	6 (2.0)	10 (2.9)	16 (2.5)
PFB	1 (0.8)	1 (0.6)	2 (0.7)	1 (0.6)	0 (0)	1 (0.3)	2 (0.7)	1 (0.3)	3 (0.5)
Total ventricular conduction defects	6 (4.5)	38 (24.2)	44 (15.2)	5 (3.0)	23 (12.2)	28 (7.9)	11 (3.7)	61 (17.6)	72 (11.2)
<i>Atrioventricular block:</i>									
1°	0 (0)	3 (1.9)	3 (1.0)	1 (0.6)	1 (0.5)	2 (0.6)	1 (0.3)	4 (1.2)	5 (0.8)
2°	0 (0)	1 (0.6)	1 (0.3)	1 (0.6)	0 (0)	1 (0.3)	1 (0.3)	1 (0.3)	2 (0.3)
<i>Arrhythmias</i>									
Unifocal ventricular extrasystoles	6 (4.5)	11 (7.0)	17 (5.9)	7 (4.2)	8 (4.2)	15 (4.2)	13 (4.4)	19 (5.5)	32 (5.0)
Multifocal ventricular extrasystoles	2 (1.5)	4 (2.5)	6 (2.1)	1 (0.6)	3 (1.6)	4 (1.1)	3 (1.0)	7 (2.0)	10 (1.6)
Repetitive ventricular extrasystoles	1 (0.8)	0 (0)	1 (0.3)	0 (0)	1 (0.5)	1 (0.3)	1 (0.3)	1 (0.3)	2 (0.3)
Total ventricular extrasystoles	8 (6.1)	15 (9.6)	23 (8.0)	8 (4.8)	11 (5.8)	19 (5.4)	16 (5.4)	26 (7.5)	42 (6.5)
Atrial or junctional extrasystoles	5 (3.8)	8 (5.1)	13 (4.5)	2 (1.2)	3 (1.6)	5 (1.4)	7 (2.3)	11 (3.2)	18 (2.8)
Atrial or junctional rhythm or tachycardia	1 (0.8)	6 (3.8)	7 (2.4)	1 (0.6)	3 (1.6)	4 (1.1)	2 (0.7)	9 (2.6)	11 (1.7)
<i>Other:</i>									
Large Q waves	1 (0.8)	2 (1.3)	3 (1.0)	0 (0)	0 (0)	0 (0)	1 (0.3)	2 (0.6)	3 (0.5)
Small Q waves	4 (3.0)	5 (3.2)	9 (3.1)	4 (2.4)	5 (2.6)	9 (2.5)	8 (2.7)	10 (2.9)	18 (2.8)
Inverted or flat T waves	4 (3.0)	14 (8.9)	18 (6.2)	8 (4.8)	17 (9.0)	25 (7.0)	12 (4.0)	31 (9.0)	43 (6.7)

*Number of electrocardiograms.

†Number in parentheses indicates percent of electrocardiograms examined (n) with each finding.

Table III. Prevalence of specific ventricular conduction defects according to age and seroreactivity to *T. cruzi*

No. of ECGs	Age (yr) 10-14		15-24		25-44		45-64		65+	
	Sero-positive (45)	Sero-negative (80)	Sero-positive (94)	Sero-negative (64)	Sero-positive (123)	Sero-negative (73)	Sero-positive (60)	Sero-negative (54)	Sero-positive (24)	Sero-negative (27)
Right bundle branch block (RBBB)	1 (2.2%)	0	6 (6.4%)	0	9 (7.3%)	0	4 (6.7%)	0	0	0
RBBB with anterior fascicular block (AFB)	0	0	3 (3.2%)	0	6 (4.9%)	0	5 (8.3%)	1 (1.9%)	2 (8.3%)	0
RBBB with posterior fascicular block (PFB)	0	0	0	0	0	0	1 (1.7%)	0	0	0
Incomplete RBBB	0	0	4 (4.3%)	1 (1.6%)	4 (3.3%)	0	1 (1.7%)	1 (1.9%)	0	0
IRBBB with AFB or PFB	0	0	1 (1.1%)	0	2 (1.6%)	0	1 (1.7%)	0	0	0
AFB	0	0	5 (5.3%)	1 (1.6%)	4 (3.3%)	1 (1.4%)	1 (1.7%)	3 (5.6%)	0	1 (3.7%)
PFB	0	0	1 (1.1%)	1 (1.6%)	0	0	0	0	0	1 (3.7%)
Total ventricular conduction defects	1 (2.2%)	0	20 (21.3%)	3 (4.7%)	25 (20.3%)	1 (1.4%)	13 (21.7%)	5 (9.3%)	2 (8.3%)	2 (7.4%)

tive women* (Table II). The prevalence rate of abnormal tracings was 4.3 times greater for seropositive men than for seronegative men, and 2.6 times greater for seropositive women than for seronegative women.

The percentage of individuals with abnormal ECGs was approximately equal for seropositive and seronegative persons in the 10 to 14 year age group, but was 9.6 times greater for seropositive persons in the 25 to 44 year age group† (Fig. 1); above 44 years this difference decreased and for those over 64 years the percentage of individuals with abnormalities was roughly equal for the seropositive and seronegative groups. Although the percentage of ECGs classified as borderline increased with age, there was little difference in these percentages between men and women or between seropositive and seronegative individuals, even when broken down according to age groups.

Multiple alterations occurred in 52.9% (37 of 70) and 55.6% (10 of 18) of the abnormal ECGs of seropositive and seronegative individuals, respectively; 28.3% of seropositive individuals with conduction defects also had extrasystoles on their tracings.

Ventricular conduction defects. Ventricular con-

duction defects (Tables II and III) were the most common alterations and occurred with age and sex distribution similar to that seen for abnormal ECGs. Left bundle branch block did not occur. Complete right bundle branch block and fascicular block with complete or incomplete right bundle branch block were strongly associated with seropositivity to *T. cruzi*.*

Complete right bundle branch block was present in 52.9% of the abnormal tracings of the seropositive group; 43.2% of these showed anterior fascicular block and 2.7% showed posterior fascicular block in addition to complete right bundle branch block. Only one seronegative individual, a 48-year-old man, had complete right bundle branch block (with anterior fascicular block). Anterior fascicular block alone occurred only 1.5 times more frequently among seropositive individuals than among seronegative individuals, but in the 15- to 44-year-old age group it was almost three times more common among seropositive persons. The total percentage of abnormal tracings with ventricular conduction defects was high in both the seronegative (61.1%) and the seropositive (87.1%) groups.

Eighty-three and a half percent of individuals with

*Chi-square test: $\chi^2 = 4.4$, $df = 1$, $p < 0.05$.

†Chi-square test: $\chi^2 = 13.0$, $df = 1$, $p < 0.001$.

*Chi-square test: complete right bundle branch block: $\chi^2 = 27.4$, $df = 1$, $p < 0.001$; combined fascicular and bundle branch block: $\chi^2 = 13.7$, $df = 1$, $p < 0.001$; isolated incomplete right bundle branch block: $\chi^2 = 2.48$, $df = 1$, $p > 0.10$; and isolated fascicular block: $\chi^2 = 0.21$, $df = 1$, $p > 0.10$.

a QRS duration above 0.09 second were seropositive (19.1% of seropositive and 4.4% of seronegative persons had a QRS interval greater than 0.09 second).*

Atrioventricular conduction defects. Complete atrioventricular block was not seen, and second-degree block occurred only twice (Table II). According to the strict criteria of the amplified Minnesota Code (PR > 0.21 second in adults and PR > 0.18 second in children under 15 with tachycardia), first-degree AV block occurred in only 1.2% of seropositive individuals and in 0.3% of seronegative individuals. However, Table IV demonstrates a strong association of seropositivity with PR intervals of 0.20 and 0.21 second in all age groups† and with intervals of 0.17 to 0.19 second for individuals under 25 years; 11.5% of seropositive individuals as opposed to 1.4% of seronegative individuals had a PR interval greater than or equal to 0.17 seconds.‡ A short PR interval (0.12 second in adults) was seen in 1.2% of seropositive individuals compared to 0.3% of seronegative individuals.

Arrhythmias. The most common arrhythmias were ventricular and supraventricular extrasystoles; ventricular tachycardia and atrial fibrillation were not seen. No difference in the prevalence rates of ventricular extrasystoles between males and females or between seropositive and seronegative individuals was noted. In the 25 to 64 year age group, ventricular extrasystoles, multifocal ventricular extrasystoles, and ventricular extrasystoles in pairs each occurred two times or more frequently among seropositive than among seronegative individuals, but the differences were not significant. Among seropositive persons, 53.8% of the ventricular ectopic activity occurred in tracings with conduction defects; only in one seropositive person were high grade (multifocal or repetitive) extrasystoles seen in an otherwise normal ECG.

The prevalence rate of atrial or junctional premature beats increased from 0.3% in the 15 to 24 year age group to 13.7% in the 65 year and over age group. The only association with seropositivity occurred in the 15 to 44 year group in which all six instances were among seropositive individuals. Sinus arrhythmia was more prevalent among seronegative than among seropositive individuals under 45 years of age.§ Neither sinus tachycardia nor sinus bradycardia were related to seroreactivity.

Other alterations. The frequency of large (Minnesota code items 1-1 and 1-2) and small (item 1-3) Q

Table IV. Association of PR interval duration, seroreactivity to *T. cruzi*, and age

PR interval duration (sec)	0.17-0.19	0.20-0.21	> 0.21
Number of individuals	44	14	4
% seropositive	65.9	85.7	100
Average age of seropositive individuals (yr)	33.8	32.8	34.0
Average age of seronegative individuals (yr)	42.8	56.0	—
% seropositive of those under 25 years	81.8	100	100

waves as recorded in Table II undoubtedly is less than that which full 12-lead tracings would detect. All large Q waves and 50% of small Q waves in the seropositive group were associated with conduction defects or arrhythmias. The average age of seropositive individuals with Q wave alterations was 43.2 years as opposed to 61.3 years in the seronegative group.

The pattern of left ventricular hypertrophy with ST segment and T wave alterations occurred only twice (in seronegative females over the age of 40). Large R waves in lead V₅ were common (29.1%) but were not associated with seroreactivity. Inverted or flattened T waves were more frequent in seropositive persons than in seronegative persons,* with the prevalence rate increasing from 5.1% in individuals under 45 years to 14.5% in individuals 45 years or older. T wave abnormalities were five times more prevalent in seropositive individuals than in seronegative individuals under 45 years, but occurred with equal frequency in individuals over 44 years of age (14% of seropositives, 15.4% of seronegatives).

Axis deviation was recorded in 9.3% of the population, 3.5 times more frequently in the seropositive group than in the seronegative group.† The most common axis deviation was left (53.3%), followed by right (30%), extreme right (15%), and indeterminate (1.7%). Other alterations including ST segment depression, low QRS voltage, and wide P waves occurred each in less than 0.5% of the population.

DISCUSSION

ECG abnormalities related to seropositivity. A high prevalence of seropositivity to *T. cruzi* and a strong association between ECG abnormalities and seropositivity were observed in this rural population in

*Chi-square test: $\chi^2 = 26.9$, $df = 1$, $p < 0.001$.

†Chi-square test: $\chi^2 = 4.29$, $df = 1$, $p < 0.05$.

‡Chi-square test: $\chi^2 = 9.8$, $df = 1$, $p < 0.01$.

§Chi-square test: under 45 years: $\chi^2 = 6.2$, $df = 1$, $p < 0.05$; under 25 years: $\chi^2 = 10.4$, $df = 1$, $p < 0.01$.

*Chi-square test: $\chi^2 = 5.2$, $df = 1$, $p < 0.05$.

†Chi-square test: $\chi^2 = 15.7$, $df = 1$, $p < 0.001$.

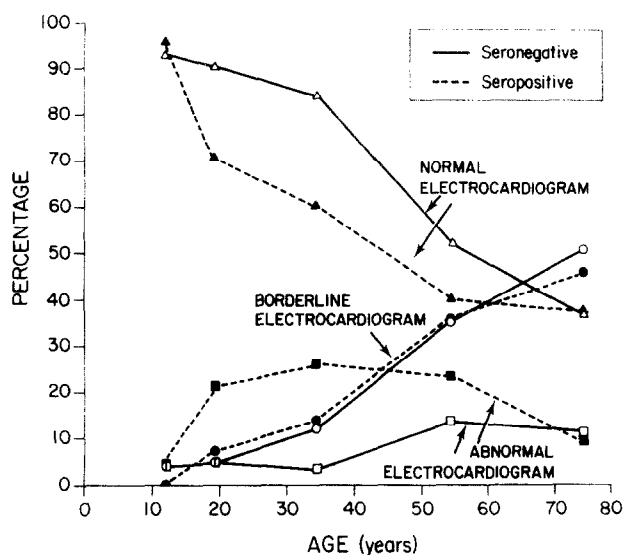


Fig. 1. Relationship of age, seroreactivity to *T. cruzi*, and ECG findings. Each point represents the percent of seropositive or seronegative individuals in each age group with normal, abnormal, or borderline ECG.

Northeast Brazil. Seropositivity to *T. cruzi* as documented by the complement fixation test has been shown to be a reliable indicator of *T. cruzi* infection.^{3, 15, 20} The ECG, however, has certain limitations. None of the ECG alterations described here are unique to Chagas' cardiomyopathy. Moreover, postmortem studies,²¹ echocardiography,²² exercise testing,²³ and His-bundle electrocardiography²⁴ have indicated cardiac disease in a small percentage of Chagasic individuals with normal resting ECGs. Nevertheless, the ECG is more sensitive than either clinical or radiologic examination in detecting cardiac pathology in Chagas' disease, particularly in asymptomatic individuals.^{10, 25} Correlation between the ECG and histologically proven lesions is often excellent, especially in the case of conduction defects.¹⁴

Gender, age, conduction defects, and extrasystoles. Our findings support the conclusions of similar studies in other geographic areas.^{2, 7, 10, 13} ECG alterations were more frequent among men than among women, despite similar age-specific rates of seroreactivity between the two groups. The peak prevalence rate of ECG abnormalities was in the 25 to 44 year age group of seropositive individuals. The frequent occurrence of conduction defects, in particular right bundle branch block with or without anterior fascicular block, is characteristic of populations in endemic areas.³ Ventricular extrasystoles were more common than atrial extrasystoles, and T wave alterations were more prevalent among young seropositive individuals than among young seroneg-

ative individuals. That the majority of infected individuals in a defined population has normal ECGs highlights the importance of the latent or indeterminate phase of Chagas' disease.²⁶

Lag period between seropositivity and ECG abnormalities. In this study, ECG abnormalities were uncommon among seropositive individuals under 15 years of age but occurred with increasing frequency in subsequent age groups up to age 45 (Fig. 1). However, in the same study area the prevalence of seropositivity increased in successively older age groups from 0% before age 1 to about 60% by age 20, and remained at this level until age 55.¹⁵ This finding suggests a lag period between primary infection and the appearance of chronic lesions; the 10 to 20 year latent phase cited from observations of individual patients is consistent with our data.^{2, 27} Similarly, Fig. 1 suggests that the acquisition of ECG abnormalities would be gradual, even within the seropositive population; indeed, limited prospective data from the few existing longitudinal studies have indicated a 1.1% per year rate of new heart disease among previously well seropositive individuals⁸ and a 2.3% per year appearance of ECG abnormalities in seropositive individuals with normal ECGs.²⁸ Despite a high prevalence of bifascicular block (complete right bundle branch block and anterior fascicular block), high-degree heart block was exceptional.

Increased mortality related to abnormal ECG. The declining prevalence rate of abnormal ECGs among older seropositive individuals suggests a higher death rate among individuals with abnormal ECGs. Selective mortality due to Chagas' heart disease could also account for the decline in seropositivity rates after 55 years observed in this population.¹⁵ Mortality statistics from other endemic areas confirm high death rates due to Chagas' disease between ages of 20 and 50.^{26, 27, 29} A retrospective analysis of a group of seropositive individuals followed over 18 years demonstrated that right bundle branch block was three times as common in fatal cases than in survivors; Chagasic individuals with atrial fibrillation and third-degree atrioventricular block had a markedly reduced life-expectancy.³⁰ In our population, a reduced prevalence of conduction defects was noted after age 65, and the absence of atrial fibrillation and complete heart block may be explained on the basis of shortened life-expectancy.

The rising percentage of individuals with borderline ECGs in successive age groups (Fig. 1) was due mainly to high frequencies of inverted or flat T waves, tall left precordial R waves, and isolated

ventricular or supraventricular extrasystoles. Such alterations are common in coronary artery disease, arterial hypertension, and old age,³¹ and in our population occurred among seronegative individuals as well as among seropositive individuals.

Interestingly, the frequency of sinus arrhythmia was significantly lower among young seropositive persons than among seronegative persons of the same age. Severe damage to the autonomic ganglia and nerves of the heart³² and disorders of autonomic control in chronic Chagasic patients³³ have been demonstrated which could account for this observation.

Strain-specific considerations. Although similar rates of seroreactivity to *T. cruzi* and ECG abnormalities have been reported from certain endemic areas,^{7,13} different rates have been described from others.¹¹ The present study was undertaken in an area where the triatomine bug *Panstrongylus megistus* is the sole domestic vector. Geographic differences in the frequency of morbidity due to Chagas' disease may be due to *T. cruzi* strain-specific characteristics or variable efficiency of transmission by different vector species. Comparative population-based epidemiologic studies using standard ECG criteria such as the Minnesota Code,¹⁸ standard serologic methods,^{15,34} and appropriate analysis should clarify observed differences among various areas.

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REFERENCES

1. Pan American Health Organization: Health conditions in the Americas 1969-1972. Pan Am Health Org Sci Pub **287**:34, 1974.
2. Laranja FS, Dias E, Duarte E, Pellegrino J: Observações clínicas e epidemiológicas sobre a moléstia de Chagas no oeste de Minas Gerais. O Hospital (Rio de Janeiro) **40**:945, 1951.
3. Laranja FS, Dias E, Nóbrega G, Miranda A: Chagas' disease, a clinical, epidemiologic and pathologic study. Circulation **14**:1035, 1956.
4. Rosenbaum MB, Cerisola JA: Epidemiologia de la enfermedad de Chagas' en la Republica Argentina. O Hospital (Rio de Janeiro) **60**:55, 1961.
5. Rosenbaum MB, Alvarez AJ: The electrocardiogram in chronic Chagasic myocarditis. AM HEART J **50**:492, 1955.
6. Dias E, Laranja FS, Nóbrega G: Doença de Chagas. Mem Inst Oswaldo Cruz **43**:495, 1945.
7. Puigbo JJ, Nava Rhode JR, Garcia Barrios H, Suarez JA, Yopez CG: Clinical and epidemiological study of chronic heart involvement in Chagas' disease. Bull WHO **34**:655, 1966.
8. Puigbo JJ, Nava Rhode JR, Garcia Barrios H, Yopez CG: A 4-year follow-up study of a rural community with endemic Chagas' disease. Bull WHO **39**:341, 1968.
9. Zeledon R, Solano G, Burston L, Swartzwelder JC: Epidemiological pattern of Chagas' disease in an endemic area of Costa Rica. Am J Trop Med Hyg **24**:214, 1975.
10. Pifano F, Anselmi A, Maekelt GA, Anselmi G, Vazquez AD: Estudios sobre la cardiopatía Chagásica en el medio rural Venezolano. Arch Venez Med Trop Parasit Med **5**:1, 1965.
11. Brant TC, Laranja FS, Bustamente FM, Melo AL: Dados sorológicos e electrocardiográficos obtidos em populações não selecionadas de zonas endêmicas de doença de Chagas no estado do Rio Grande do Sul. Rev Bras Malariol Doencas Trop **9**:141, 1957.
12. Porto CC: O eletrocardiograma no prognóstico e evolução da doença de Chagas. Arq Bras Cardiol **17**:313, 1964.
13. Lucena DT, Costa EG, Cordeiro E: Alterações electrocardiográficas na doença de Chagas no nordeste do Brasil. Rev Bras Malariol Doencas Trop **15**:369, 1963.
14. Andrade ZA, Andrade SG, Oliveira GB, Alonso DR: Histopathology of the conducting tissue of the heart in Chagas' myocarditis. AM HEART J **95**:316, 1978.
15. Mott KE, Lehman JS, Hoff R, Morrow RH, Muniz TM, Sherlock I, Draper CC, Pugliese C, Guimarães AC: The epidemiology and household distribution of seroreactivity to *Trypanosoma cruzi* in a rural community in northeast Brazil. Am J Trop Med Hyg **25**:552, 1976.
16. Maekelt GA: Die Komplementbindungsreaktion der Chagaskrankheit. Z Tropenmed Parasitol **11**:152, 1960.
17. Maguire JH, Mott KE, Almeida EC, Ramos NB, Souza JA, Guimarães AC: Electrocardiographic classification and abbreviated lead system for population-based studies on Chagas' disease. Bull Pan Am Health Organ **16**:47, 1982.
18. Rose, GA, Blackburn H: Cardiovascular survey methods. WHO Monograph Series No. 56, Geneva, 1968, World Health Organization, 188 pp.
19. New York Heart Association, Criteria Committee: Nomenclature and criteria for diagnosis of diseases of the heart and great vessels. 8th ed. Boston, 1979, Little, Brown, & Company, 349 pp.
20. Rassi A, Amato Neto V, Siqueira AF: Comportamento evolutivo da reação de fixação do complemento na fase crônica da moléstia de Chagas. Rev Inst Med Trop Sao Paulo **11**:430, 1969.
21. Pinto Lima FX, Spiritus O, Tranchesi J: Arrhythmias and vector electrocardiographic analysis of complete bundle branch block in Chagas' disease. A study of 103 autopsied cases. AM HEART J **56**:501, 1958.
22. Palermo HA, Caeiro TF, Iosa DJ: Características distintivas de la cardiopatía en la enfermedad de Chagas. Medicina (Buenos Aires) **40**(suppl 1):234, 1980.

23. Pereira MHB, Brito FS, Levi G, Pereira CG, Lion MF, Amato Neto V: Teste ergométrico (TE) em portadores de doença de Chagas "forma indeterminada." Estudo de 20 casos. *Arq Bras Cardiol* **29**(suppl 1):89, 1976.
24. Gruppi C, Pileggi F, Sosa E, Bellotti G, Carmargo PR, Garcia DP, Decourt LV: Eletrograma do feixe de His (EFH): Estudo da condução atrioventricular (AV) com estimulação atrial em pacientes sem cardiopatia com Machado-Guerreiro (MG) positivo. *Arq Bras Cardiol* **29**(suppl 1):234, 1976.
25. Alvarez AJ, Rosenbaum MB: Radiologia cardiovascular en la miocarditis crónica Chagásica. *Rev Argent Cardiol* **20**:146, 1953.
26. Prata AR: Natural history of Chagasic cardiomyopathy. *Pan Am Health Org Sci Pub* **318**:191, 1976.
27. Dias JCP: Epidemiological aspects of Chagas' disease in the Western (sic) of Minas Gerais, Brasil. Environmental, ecological and human aspects studied by the Bambui Center (FIOCRUZ) during the period 1943-1976. Round table presentation in Abstracts of Congresso Internacional sobre Doença de Chagas, Rio de Janeiro, 1979, p. H1.
28. Macedo V, Prata A: Forma indeterminada da doença de Chagas. XIV Cong Soc Brasil Med Trop e III Cong Soc Brasil Parasit Joao Pessoa, 1978, Resumos p 32.
29. Puffer RR, Griffith GW: Patterns of urban mortality. Report of the InterAmerican investigation of mortality. *Pan Am Health Org Sci Pub* **151**:139, 1967.
30. Dias JCP, Kloetzel K: The prognostic value of the electrocardiographic features of chronic Chagas' disease. *Rev Inst Med Trop Sao Paulo* **10**:158, 1968.
31. Marriott HJL: Practical electrocardiography. 5th ed. Baltimore, 1972, The Williams & Wilkins Company, 325 pp.
32. Chapadeiro E, Lopes ER, Pereira FEL: Denervação parasimpática e hipertrofia do miocárdio em chagásicos crônicos. *Rev Inst Med Trop Sao Paulo* **9**:40, 1967.
33. Amorim DS, Manço JC, Gallo L, Marin Neto JS: Clínica: Forma crônica cardíaca. In Brener Z, Andrade Z, editors: *Trypanosoma cruzi e Doença de Chagas*. Rio de Janeiro, 1979, Guanabara-Koogan, p 265.
34. Almeida JO, Fife EH Jr: Quantitatively standardized complement fixation methods for critical evaluation of antigens prepared from *Trypanosoma cruzi*. *Pan Am Health Org Sci Pub* **319**:86, 1976.