CHAGAS DISEASE - 2006

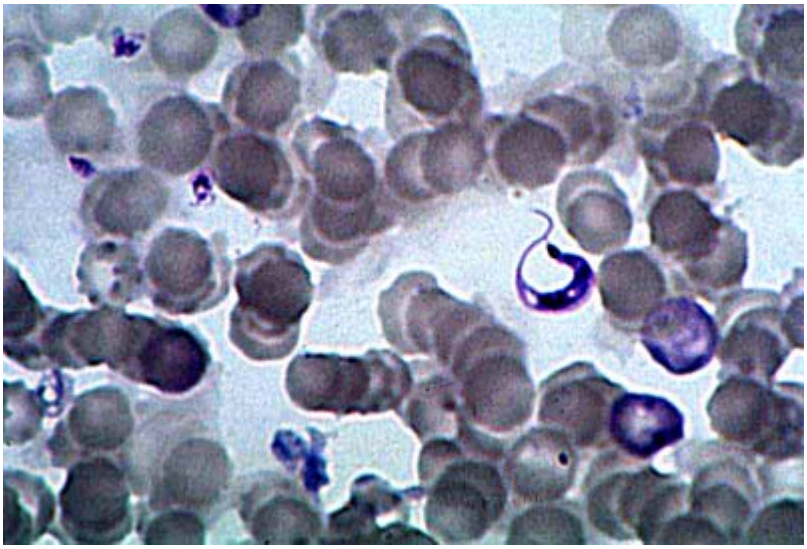
Dr. Andrés R. Pérez Riera

Chagas' disease, is caused by the protozoan parasite Trypanosoma cruzi (Figures1 and 2).

The entity affects 16- 20 million people in the Americas are infected (**Anon. *Tropical disease research progress 1975–1994 highlights 1993–1994. Twelfth programme report of the UNDP/World Bank/WHO Special Programme for Research and Training in Tropical Diseases (TDR)*. Geneva: World Health Organization, 1995:125–34.**), and that more than 100 million are at risk. Of those infected, 45,000 - 50,000 will die each year. Mortality is mainly due to chronic chagasic cardiomyopathy. Sudden death, usually due to ventricular fibrillation, is the principal cause of death in 60% of cases. Bradyarrhythmia, thromboembolic phenomena, and, rarely, a ruptured aneurysm, are other causes of sudden death. Congestive heart failure (25-30% of cases), cerebral or pulmonary embolism (10-15% of cases). Symptomatic acute phases mainly occur in newborns (congenital infection) or young children. Chagasic esophagopathy is observed more frequently in the second decade of life, and chronic chagasic cardiomyopathy and colopathy are generally detected later, in the third, fourth, or fifth decade of life.

Figure 1

Figure 2

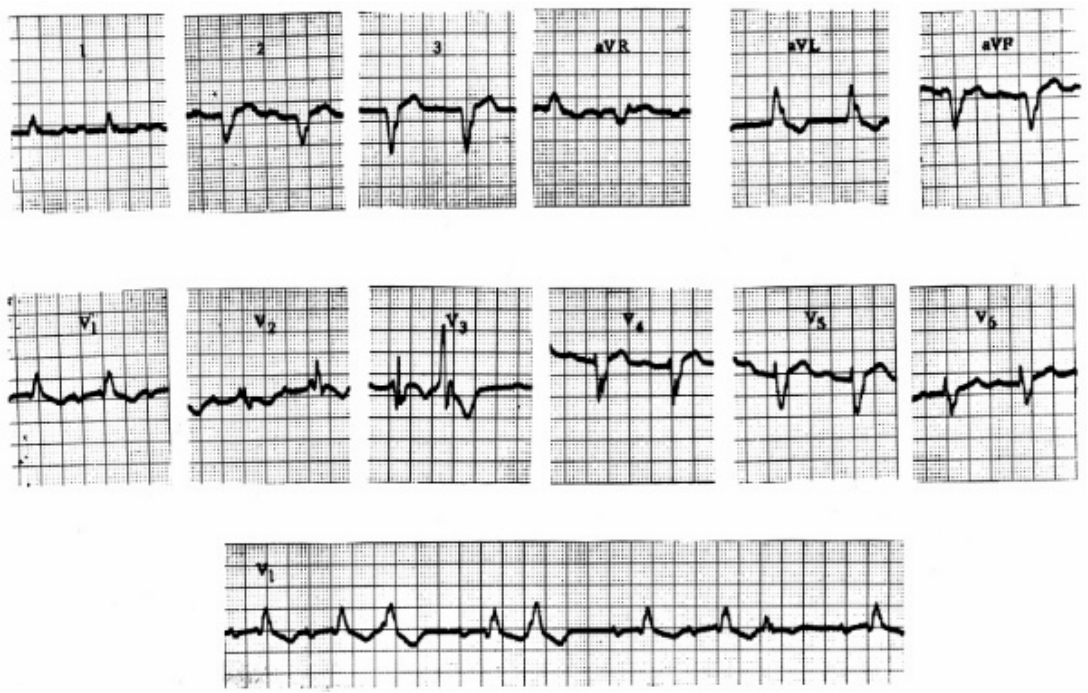


There are 3 stages in the disease **Elizari M. La miocardiopatía chagásica: perspectiva histórica. *Medicina* 1999; 59(suppl II):25–40.**:

1) **The acute phase**, lasting from 1-3 months, The inflammation of the acute phase of Chagas' myocarditis, as seen in one single case, did not seem to interfere with conduction through the AV system;
2) **The latent or intermediate phase**, which may last from 10-20 years, and;
3) **Chronic phase**, which has the most serious manifestations. Köberle and others believed that the cardiomegaly is caused by the same basic disturbance of intrinsic innervation as has been demonstrated in the alimentary tract. Most pathologists agree there is a marked reduction in identifiable ganglion cells in the heart. Microscopically, there is a diffuse inflammation of the myocardium involving the walls of all chambers, including the septum, and sometimes extending into the endocardium and epicardium. The valves and coronary arteries are not affected. In chronic Chagas' myocarditis the conducting tissue showed extensive and variable changes: chronic inflammation, fibrosis, atrophy and fragmentation of specific fibers, extreme dilatation and tortuosity of veins, capillaries and lymphatics, fatty infiltration, and arterial medial and intimal fibrosis. On physical examination, there may be cardiac enlargement, irregularities of rhythm due to premature ventricular contractions (PVCs) or atrioventricular block, gallop rhythm, sinus bradycardia, diminished heart sounds, a widely split second pulmonary sound, and systolic murmurs from functional mitral or tricuspid regurgitation. There may also be an increase in systemic venous pressure and hepatomegaly. The systolic blood pressure is usually normal to moderately lowered, as is the pulse pressure. A preferential involvement of the right bundle branch and the anterior fascicle of the left branch was observed and an excellent correlation with

electrocardiographic abnormalities was found. There was also evidence presented that bundle branch block may be caused by disease proximal to the bundle branches. Complete AV block seemed to be the final result of the progressive inflammatory and degenerative changes involving the conduction system in chronic Chagas' myocarditis. Inflammation and fibrosis did also involve the SA node, Purkinje fibers, intracardiac nervous ganglia, and the contractile myocardium. **Andrade ZA, Andrade SG, Oliveira GB,et al. Histopathology of the conducting tissue of the heart in Chagas' myocarditis. Am Heart J. 1978;95:316-324.** The Figure 3 show a typical ECG

Figure 3



Typical ECG in chronic Chagas' cardiomyopathy is often diagnostic, showing various conduction disturbances, principally right bundle branch block and left anterior fascicular block. Negative T waves of the primary type and alterations indicative of apical

myocardial necrosis are also common. Frequent and multifocal PVCs are characteristic, as is a sinus bradycardia or an abnormally fixed sinus rhythm associated with heart failure.

The polymorphism of the clinical manifestations(**Puigbó J, Giordano H, Suárez C, *et al.* Aspectos clínicos en la enfermedad de Chagas. In: Madoery R, Madoery C, Cámera M, eds. *Actualizaciones en la enfermedad de Chagas. Simposio satélite. Organismo oficial del Congreso Nacional de Medicina.* 1993:27–38.**) can be summarised as follows: intraventricular conduction system abnormalities, ventricular arrhythmia, sinus node dysfunction, left ventricular segmental lesions, and enlargement and dysfunction of left ventricle with or without heart failure. The main causes of death are heart failure and sudden death (about 70% and 30%, respectively). (**Manzullo EC, Chuit R. Risk of death due to chronic chagasic cardiopathy. *Mem Inst Oswaldo Cruz* 1999;94(suppl 1):317–20.**)

Since segmental abnormalities were predominantly observed in apical and para-apical areas of the ventricles, performance of right and left cineventriculograms is recommended before implantation of cardiac pacemakers. **Carrasco HA, Medina M, Inglessis G et al. Right ventricular function in Chagas disease. Int J Cardiol. 1983; 2:325-338**.

The diversity and severity of the symptoms range from a mild electrocardiographic alteration to sudden death. The electrocardiographic alterations are usually the first evidence of disease progression. During the past decades, after urban migrations, Chagas disease became frequent in cities and a health problem in non-endemic countries, where it can be transmitted vertically and by blood transfusion or organ transplantation. Microepidemics of acute Chagas disease have been reported, probably due to oral transmission. Heart involvement is the major feature of

the disease because of its characteristics, frequency, and consequences, and is also the source of most controversies. **Prata A. Clinical and epidemiological aspects of Chagas disease. Lancet Infect Dis. 2001;1:92-100.**
The indeterminate form of Chagas' disease clearly represents a benign condition with a favorable long-term prognosis. Although some patients develop electrocardiographic changes, left ventricular systolic function is well preserved. **Ianni BM, Arteaga E, Frimm CC, Chagas' heart disease: evolutive evaluation of electrocardiographic and echocardiographic parameters in patients with the indeterminate form. Arq Bras Cardiol. 2001; 77:59-62**.
In asymptomatic chagasic patients an abnormal ECG is observed in almost 20% of cases and Left anterior fascicular block the most frequent alteration found. **Rigou DG, Gullone N, Carnevali L, Asymptomatic Chagas disease. Electrocardiographic and echocardiographic findings Medicina (B Aires). 2001;61:541-544.)(Rigou DG,High prevalence of left anterior hemiblocking in the electrocardiogram in asymptomatic Chagas cardiomyopathy Medicina (B Aires). 2001;61:114-115.).**
A narrow QRS does not exclude the presence of significant global and segmental LV dysfunction in Chagasic patients. **Ribeiro AL, Rocha MO, Barros MV A narrow QRS does not predict a normal left ventricular function in Chagas' disease. Pacing Clin Electrophysiol. 2000;23:2014-2017**

Right-branch bundle blocks (RBBB), PVCs and left anterior fascicular block (LAFB) are the commonest cardiac arrhythmias among the chagasic patients, and each of these types of arrhythmia (alone or with other types of arrythmia) are more frequent in the chagasic patients than the non-chagasic. The incidence of RBBB among the arrhythmic varied significantly with age in the non-chagasic patients (increasing with age among both the males and females) but not among the chagasic subjects. When the

frequencies of each type of arrythmia and each combination of types is compared, the co-occurrence of RBBB and another type of arrhythmia are almost indicative of American trypanosomiasis (occurring in 30.6% of the chagasic subjects but only 2.6% of the non-chagasic). Similarly, only 0.4% of the non-chagasic patients but 7.4% of the chagasic have RBBB, VE and LAFB concurrently. However, the frequencies of RBBB in isolation (i.e. with no other, concurrent, electrocardiographic abnormality), VE in isolation, or LAFB in isolation are not significantly different in the chagasic and non-chagasic patients. **Jorge MT, Macedo TA, Janones RS, Types of arrhythmia among cases of American trypanosomiasis, compared with those in other cardiology patients. Ann Trop Med Parasitol. 2003; 97:139-148**

QT interval parameters are potential prognostic markers of arrhythmogenicity risk and cardiovascular mortality The QT-interval dispersion (QTd) and left ventricular (LV) end-systolic dimension are the strongest independent mortality predictors in patients with Chagas' disease. Heart rate, the presence on ECG of pathological Q waves, frequent PVCs, and isolated LAFB refined the mortality risk stratification. **Salles G, Xavier S, Sousa A, et al. Prognostic value of QT interval parameters for mortality risk stratification in Chagas' disease: results of a long-term follow-up study. Circulation. 2003;108:305-312**.

The involvement of the autonomic nervous system in Chagas' disease has been the subject of many studies, which have become more numerous since Koberle's pioneering work in the 1950s, showing the partial or total destruction of cardiac neurons. Parasympathetic dysautonomia may be an early phenomenon (patients at an early stage of cardiac involvement) and may precede left ventricular systolic dysfunction thus, the indices that reflect parasympathetic activity (pNN50 and rMSSD) are increased in Chagas patients. **Cunha AB, Cunha DM, Pedrosa RC, et al.Norepinephrine and heart rate variability: a marker**

of dysautonomia in chronic Chagas cardiopathy. Rev Port Cardiol. 2003; 22:29-52.
Twenty-four-hour Holter monitoring and heart rate variability allow to establish differences between the healthy subjects and patients with Chagas infection without evidence of cardiac disease.
Heart rate turbulence (HRT) It parameter quantifies the biphasic response of the sinus node to VPVCs) and is a powerful ECG related risk predictor. PCs are frequent in Chagas disease, a potentially lethal illness, and can hamper the analysis by conventional methods of autonomic heart control. Ribeiro et al. examined HRT in patients with Chagas disease. Chagas disease patients and healthy controls (group 0, n = 11) without other diseases were submitted to a standardized protocol, including ECG, echocardiography, and 24-hour Holter monitoring. Chagas disease patients were divided according to their left ventricular systolic function: normal (group 1, n = 103) and reduced ejection fraction (group 2, n = 23). Two HRT indices, turbulence onset (TO) and turbulence slope (TS), were calculated and compared among groups after adjustment for covariates like the prevalence of VPCs and the mean heart rate. Chagas disease patients had significantly altered TO and TS values in comparison with controls for both comparisons. The authors conclude that, HRT data may be useful in the ECG analysis of autonomic heart control in Chagas disease. **Ribeiro AL, Schmidt G, Sousa MR, Heart rate turbulence in Chagas disease. Pacing Clin Electrophysiol. 2003;26:406-410**. The physiopathological and clinical significance of denervation in Chagas disease is still incompletely understood.
Although myocardial infarction is most often the manifestation of epicardial coronary artery disease, Chagas heart disease due to chronic Trypanasome Cruzi infection may present with a syndrome of chest pain and elevations in markers of cardiac myonecrosis. In the setting of an increasingly diverse global population and immigration of peoples from endemic areas of Trypanasome Cruzi, it is

important to be aware of the myriad cardiac manifestations of Chagas disease. **ElMunzer BJ, Sallach SM, McGuire DK. Cardiac chagas disease masquerading as an acute myocardial infarction. Cardiol Rev. 2004; 12:69-72**.

At the present time the assessment of results of treatment of Chagas disease is mainly parasitological. Anti Trypanosoma cruzi IgGs remain positive practically lifelong and electrocardiographic tracings are not usually used as criteria of improvement. There is no association between the persistence of the parasite in treated patients with Chagas disease and the evolution of electrocardiographic tracings. **Zulantay I**, **Arribada A**, **Honores P**, **Sanchez G**, et al. **No association between persistence of the parasite and electrocardiographic evolution in treated patients with Chagas disease Rev Med Chil. 2005; 133:1153-1160**.

Ventricular tachycardia (VT) is common among patients with Chagas' heart disease but the ultimate mechanisms responsible for its sustained and nonsustained forms are not understood. **Sarabanda AV, Sosa E, Simoes MV, Ventricular tachycardia in Chagas' disease: a comparison of clinical, angiographic, electrophysiologic and myocardial perfusion disturbances between patients presenting with either sustained or nonsustained forms. Int J Cardiol. 2005;102:9-19**.

1) VT may arise from various regions in both ventricles, but LV inferolateral scar is the main source of S-VT reentrant circuits;
2) There is good topographic correlation between myocardial perfusion, wall motion abnormalities and areas that originate S-VT;

3) Although to a lesser extent, wall motion and perfusion defects also occur in a relevant proportion of chagasics with NS-VT.
4) Syncope episodes and induction of S-VT by programmed ventricular stimulation are significantly more frequent in S-VT patients.
5) A significantly higher prevalence of wall motion abnormalities (S-VT: 82% versus NS-VT: 46%, p = 0.005) and myocardial perfusion defects (basal segments, S-VT: 95.5% versus NS-VT: 44%, p = 0.001) was documented within the LV inferior and/or posterolateral regions in S-VT patients compared to NS-VT.
6) Induction of SMVT during programmed ventricular stimulation is a predictor of arrhythmia occurrence cardiac death and general mortality in patients with chronic chagasic cardiomyopathy and NS-VT. **Silva RM, Tavora MZ, Gondim FA, Predictive value of clinical and electrophysiological variables in patients with chronic chagasic cardiomyopathy and nonsustained ventricular tachycardia. Arq Bras Cardiol. 2000;75:33-47**.
7) The mechanism of VT is likely to be reentrant in many patients, and therefore VT can be produced by extrastimuli. Electrophysiologic study is therefore useful for establishing the diagnosis of S-VT and may be useful for guiding initial therapy in selected cases of Chagas' disease. **Mendoza I, Camardo J, Moleiro F,** et al.**Sustained ventricular tachycardia in chronic chagasic myocarditis: electrophysiologic and pharmacologic characteristics. Am J Cardiol. 1986;57:423-427**.
8) Under electrophysiologic studies, 44 asymptomatic chagasic individuals were studied by Pimenta el col. The mean age was 39.9 years, with 26 being male. The surface ECG showed normal tracings in 12 patients, LAFB in three, IRBBB in two, and CRBBB in 27 (isolated in six, associated with LAFB in 19 and

with LPFB in two). Sinus nodal dysfunction was found in eight (18.1%), and 29 (66%) presented with some form of abnormal AV nodal behavior during atrial stimulation. Repetitive ventricular beats were recorded in 17 (41.4%) patients. In 19 (43.1%), patterns of dysfunction at two or more levels of the cardiac conducting system were found. It is concluded that the chagasic cardiomyopathy produces diffuse lesions in the conducting tissue of the human heart in the stages when the disease is detected, and that the individuals are still asymptomatic. **Pimenta J, Miranda M, Pereira CB. Electrophysiologic findings in long-term asymptomatic chagasic individuals. Am Heart J. 1983;106:374-380**.

Complete atrioventricular block may be another clinical manifestation of Trypanosoma cruzi infection reactivation in Chagas' heart transplant recipients. **Bestetti RB, Cury PM, Theodoropoulos TA,et al.Trypanosoma cruzi myocardial infection reactivation presenting as complete atrioventricular block in a Chagas' heart transplant recipient. Cardiovasc Pathol. 2004;13:323-326.**
Kiss of the Benchuca--Chagas' disease presenting with transient third-degree atrioventricular block.

Cardiac resynchronization therapy (CRT) combined with implantable cardioverter defibrillator (ICD) from the outset may be recommended in patients with permanent atrial fibrillation and severe dilated cardiomyopathy due to Chagas disease **(da Silva Menezes A. Outcome of right ventricular bifocal pacing in patients with permanent atrial fibrillation and severe dilated cardiomiopathy due to Chagas disease: three years of follow-up. J Interv Card Electrophysiol. 2004;11:193-198.)**

Indicators of progression in early-stage Chagas' heart disease(patients without heart failure) **Viotti R**, **Vigliano C**, **Lococo B**, **Clinical predictors of chronic chagasic myocarditis progression** Rev Esp Cardiol. 2005;58:1037-1044.

1) Age at entry;
2) Left ventricular systolic diameter;
3) Left ventricular (LV) systolic dysfunction is a major prognostic determinant in Chagas' disease
4) Intraventricular conduction abnormalities;
5) Sustained ventricular tachycardia;
6) Male gender has higher risk of progression to chronic Chagas cardiomyopathy. **Basquiera AL**, **Sembaj A**, **Aguerri AM**, **Risk progression to chronic Chagas cardiomyopathy: influence of male sex and of parasitaemia detected by polymerase chain reaction. Heart. 2003; 89:1186-1190**.

Treatment with benznidazole reduced the risk of progression.

Specific clinical indicators and a derived clinical risk score can be used to predict the progression of chronic chagasic myocarditis in patients without heart failure.

The T-wave axis shift has been reported to represent a general marker of ventricular repolarization abnormalities and a potential indicator of increased risk for cardiovascular mortality. The frontal plane T-wave axis was estimated by Souza et al from 12-lead electrocardiograms obtained on admission and categorized as:

1) Normal (+15 degrees to +75 degrees);
2) Borderline (+75 degrees to +105 degrees or +15 degrees to -15 degrees
3) Abnormal (>105 degrees or < -15 degrees).

Kaplan-Meier survival curves showed that the 3 categories of T axis had significantly different prognoses. Multivariate Cox's survival analysis demonstrated that an abnormal T axis increases the risk of death threefold and sudden death nearly sixfold after adjustment for other covariates,

including left ventricular systolic function and other electrocardiographic abnormalities. Borderline T-wave axis also indicated a worse prognosis, particularly in the subgroup of patients with abnormal baseline electrocardiograms.
T-wave axis deviation is an easily quantified, strong, and independent mortality risk predictor in patients with chronic Chagas' disease. **Salles GF, Xavier SS, Sousa AS, T-wave axis deviation as an independent predictor of mortality in chronic Chagas' disease. Am J Cardiol. 2004;93:1136-1140.**

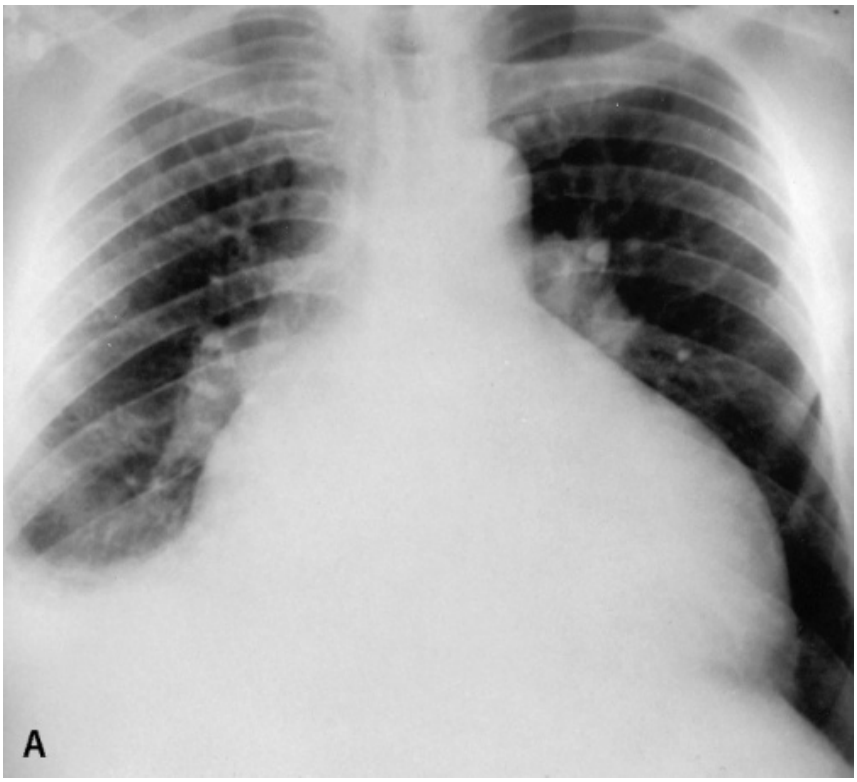
High parasitemia do not have an influence on the evolution of the chronic infection. **Castro C, Prata A, Macedo V.The influence of the parasitemia on the evolution of the chronic Chagas' disease Rev Soc Bras Med Trop. 2005;38:1-6**

Imaging Diagnosis

In 20% of patients with severe chronic Chagas' myocardiopathy, the cardiac size is normal or only slightly enlarged. Equally, some patients who have severe heart disease clinically with cardiomegaly and advanced atrioventricular block have few or no symptoms. There is good correlation between the intensity of the microscopic changes and the degree of cardiac enlargement. The size of the heart depends on the severity and location of myocardial lesions and varies from normal to enormous dilatation and hypertrophy.

In the early stages of heart failure, isolated dilatation of the left ventricle may be present, since damage there is usually more severe than in other chambers and it has a heavier work load. Loss of distensibility of the left ventricular wall due to marked fibrosis may cause annular dilatation and functional incompetence of the mitral valve, and restricted diastolic filling of the left ventricle with resultant pulmonary venous congestion. Dyspnea is then more marked and on

chest x-rays the cardiac contour, lung fields and hila resemble the radiographic appearance of mitral and aortic valvular disease rather than a myocardiopathy (**Figure 4.**). At times, the left atrium may be quite prominent and an apical systolic murmur may be present, imitating mitral insufficiency.



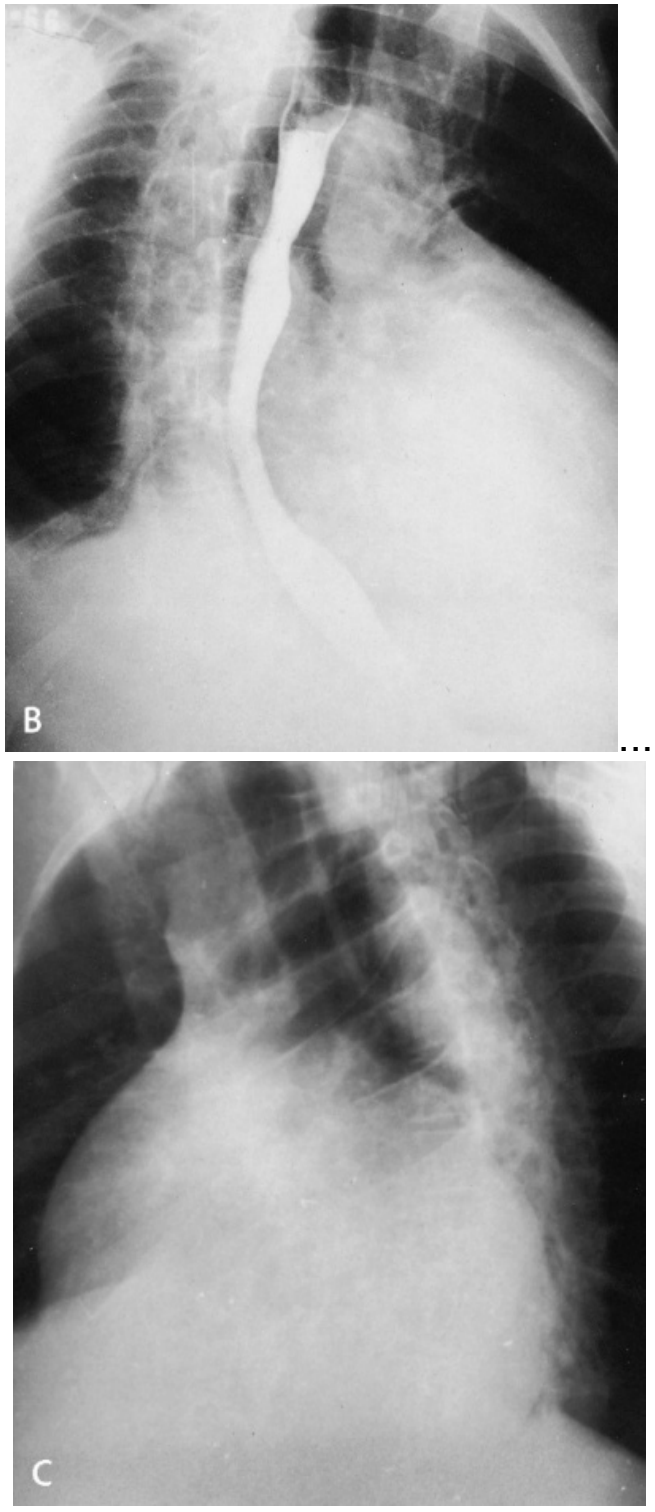


Fig. 4. Chronic Chagas' myocardiopathy in a 55-year-old black Brazilian man. PA (**A**), right anterior oblique with barium in the esophagus (**B**), and left anterior oblique views of the chest (**C**) show marked generalized cardiac enlargement with prominence especially of the left ventricle and left atrium. The latter causes a prominent indentation upon the barium-filled esophagus in the RAO view, and also

elevation of the left main stem bronchus and straightening of the left heart border on the PA view. There is minimal pulmonary vascular congestion with some cephalization of venous blood flow. There is also dilatation of the superior vena cava and a small right pleural effusion. In such patients, loss of distensibility of the left ventricular wall from fibrosis may cause functional mitral valve incompetence and restricted diastolic filling of the left ventricle, with resultant pulmonary venous congestion. The cardiac silhouette, pulmonary vasculature and hilar areas may resemble the radiographic appearance of mitral and/or aortic valvular disease rather than a myocardiopathy. Clinically, this patient had shortness of breath, easy fatiguability, an enlarged liver, and weight gain for the past year. The heart sounds were weak with a few premature beats. No murmurs were heard. EKG showed slight sinus tachycardia, complete right bundle branch block, left ventricular hypertrophy, a wide QT interval and isolated premature ventricular contractions. Serologic reaction for Chagas' disease (Machado-Guerreiro test) was positive. Necropsy showed Chagas' cardiopathy with a heart weight of 650 gm, generalized cardiac enlargement, right ventricular infarction, chronic passive congestion of the viscera, hydrothorax and ascites, and partial denervation of the esophagus and Auerbach plexi of the sigmoid colon. (Courtesy of Dr. Clovis Simao, Sao Paulo).

In the usual case of simultaneous left- and right-sided failure, signs of pronounced systemic venous hypertension overshadow those of pulmonary venous congestion. Dyspnea and orthopnea are not severe even in advanced disease. The chest radiograph typically shows prominent generalized cardiomegaly, but clear pulmonary fields without congestion and small to normal hila even when there is severe cardiac failure (**Figs 5** and **6**).

The main pulmonary artery segment and peripheral vasculature are usually normal, probably because of the failure of the right ventricle to overload the lungs. In many patients with advanced congestive failure (**Fig. 7**), predominant right-sided failure with functional tricuspid insufficiency may develop.

Figs 5 and **6**

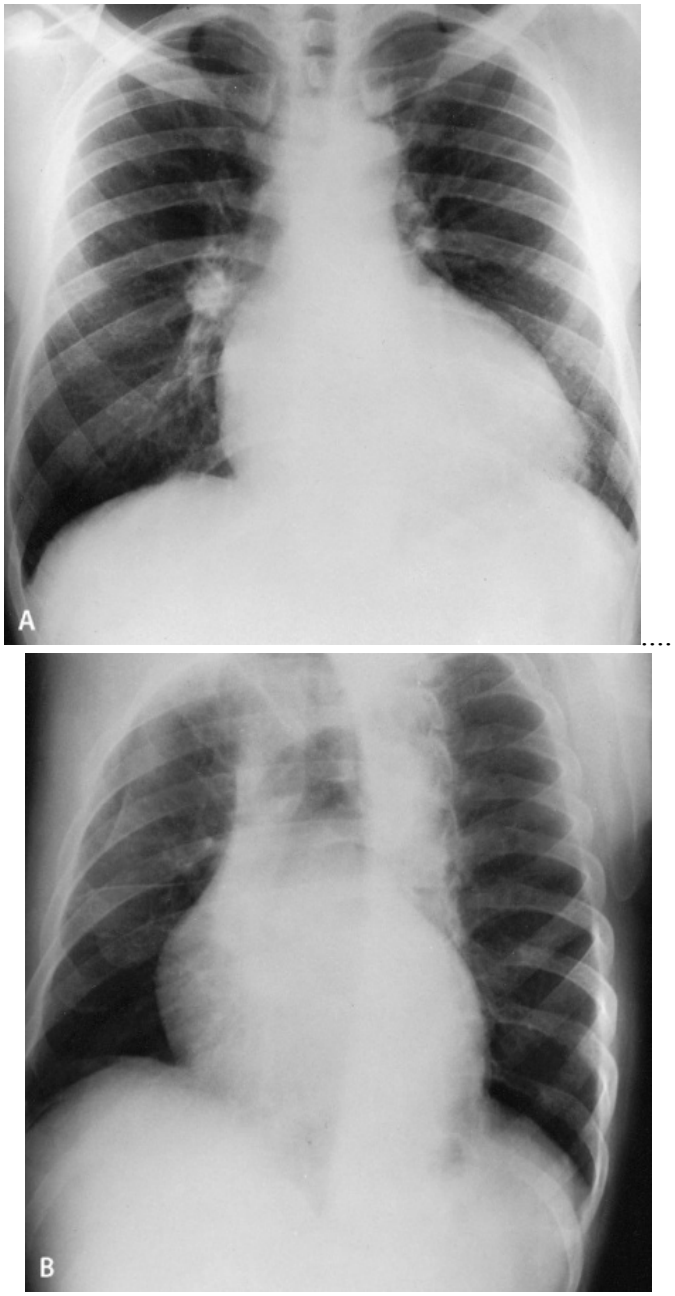


FIGURE 7

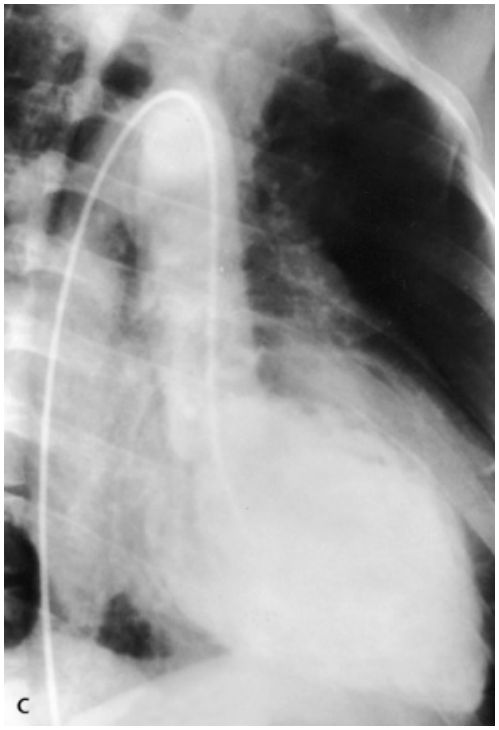
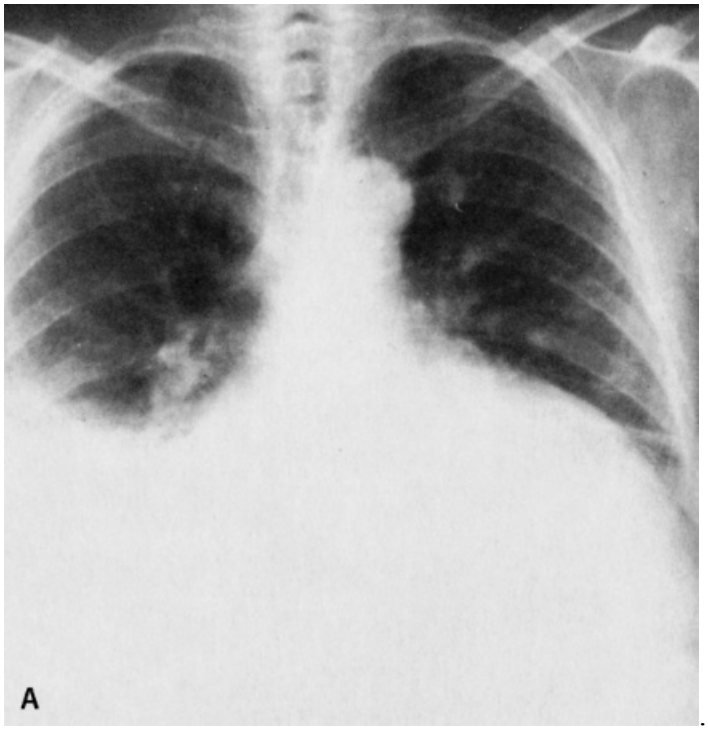


Fig.7 Chronic Chagas' myocardiopathy in an adult Brazilian man. (**A**) PA and (**B**) left anterior oblique views of the chest show moderate cardiac enlargement with prominence especially of the left ventricle. Note also the clear lung fields with no pulmonary congestion and no pleural effusion. Following retrograde passage of a catheter through the thoracic aorta and into the left ventricle, subsequent injection of contrast media shows an enlarged left ventricular chamber with characteristic aneurysm formation at the apex (**C**). (Courtesy of Dr. Clovis Simao).



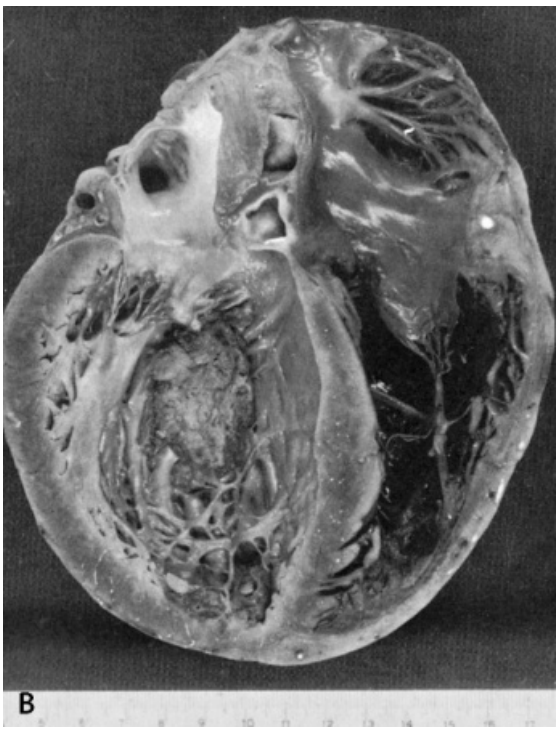


Fig.8 Chronic Chagas' cardiomyopathy in a 57-year-old white Brazilian woman with advanced biventricular failure. PA view of the chest (**A**) shows marked cardiac enlargement, predominantly left ventricular, with pulmonary vascular congestion and a large right pleural effusion. The patient complained of shortness of breath of 2 years duration, progressively increasing in the past 2 months. She had two pillow orthopnea, pitting edema of the lower

extremities and facial edema. Heart sounds were weak and there was an arrhythmia, but no murmurs. EKG showed left bundle branch block and multifocal ventricular extrasystoles. Chagas' serology was positive. At necropsy there was a generalized cardiac enlargement and hypertrophy with a heart weight of 550 gm and mural thrombosis in the left ventricle (**B**). There was evidence of congestive heart failure with hydrothorax, chronic liver congestion, and lower leg edema. AFIP 69-2960. (Courtesy of Dr. Clovis Simao).

Echocardiography is today the best imaging modality in the evaluation of chronic Chagas' myocardiopathy. The findings basically reflect cardiac dilatation and compromise of heart function. The abnormalities seen on bidimensional echocardiography include:

1) Dilatation and diffuse myocardial involvement, which characterize this myocardiopathy, with an increased volume/mass ratio;

2)Increase in left ventricular systolic and diastolic volume;

3) Septal and posteroinferior wall hypokinesis; (4) segmental hypokinesis of the posterior wall of the left ventricle;

5) Diffuse hypokinetic pattern (observed in up to 25% of patients) and;

6) Decrease in ventricular function with reduced ejection.

Cineangiography

Carrasco et al (**Carrasco HA, Barboza JS, Inglessis G, et al Left ventricular cineangiography in Chagas' disease: detection of early myocardial damage. Am Heart J. 1982;104:595-602.**) used cineangiography to identify left ventricular signs of myocardial damage and study the evolution of characteristic lesions in 126 chronic chagasic patients, divided into 3 groups.

Group I: Patients had no clinical, ECG or radiological evidence of heart disease; 41% of them showed apical or anterior apical asynergy, suggesting early subclinical myocardial damage.

Group II: Patients had an abnormal ECG but no clinical signs of heart failure. Extensive asynergy, LV dilatation, decreased distensibility and contractility were present in 98% of these patients.

Group III: Patients had congestive heart failure, a greatly dilated, hypokinetic left ventricle and a 40% incidence of large apical aneurysms; 20% had thombosis within the LV.

These investigators concluded that left cineventriculography enabled them to diagnose early myocardial damage and detect potentially resecable lesions such as ventricular aneurysms and apical thromboses.

THE CAUSES OF LEFT VENTRICULAR ANEURYSM WITHOUT CORONARY ARTERY DISEASE

1) Myocarditis;
2) Arrhythmogenic right ventricular dysplasia;
3) Chagas' disease;
4) Glycogen storage disease;
5) Sarcoidosis.

Left ventricular aneurysm formation without coronary artery disease is a rare phenomenon. Ventricular tachycardia is a significant complication in these patients. In first Word the main cause is hypertrophic cardiomyopathy (HCM),in dilated phase and or midventricular type. I South America is Chagas disease. This serious disease usually does not develop until years after infection and is characterized by chronic inflammation, tissue damage and fibrosis. In this particular heart, the fibrosis at the apex of the heart was so severe that the muscle wall became thin and an aneurysm, or a "ballooning-out," developed. A penlight was placed in the heart to illuminate this aneurysm.

Endomyocardial fibrosis and thinning occur, particularly at the apex of the left ventricle, where aneurysmal dilatation may develop in over 50%, and in some reports as many as 86%, of patients who come to autopsy (**Fig. 3**). Possible mechanisms for development of these apical aneurysms include ischemia, inflammation, herniation through the apical spiral bundles, or autonomic imbalance. There are frequently mural thrombi in the aneurysm or the apex of the ventricles or attached elsewhere to the endocardium of the atria or ventricles.

Fig. 3
APICAL ANEURYSMS IN CHAGASIC HEART