

## ACUTE MYOCARDIAL INFARCTION IN CHRONIC CHAGAS' CARDIOMYOPATHY. REPORT OF TWO CASES WITH NO OBSTRUCTIVE CORONARY ARTERY LESIONS

Silvia G. LAGE, Antonio P. MANSUR, José A. F. RAMIRES, Protásio da LUZ, Giovanni BELLOTTI & Fúlvio PILEGGI

### S U M M A R Y

This report describes two patients with chronic Chagas' Heart Disease who developed clinical and laboratorial signs of myocardial infarction. Both patients presented sudden oppressive chest pain, without precipitating factor. In the first case, the highest MB-CK value was 65 IU, 22 hours after the beginning of the pain. On the second case, it was 77 IU at 18 hours after the beginning of the pain. In both cases ECG changes suggesting non-transmural infarction were present. The  $^{99m}\text{Tc}$  PYP myocardial scintigram of the first case was positive. Coronary angiograms performed on the 18th and 9th day, respectively, after the acute infarction did not display obstructive lesions. Possible mechanisms causing myocardial infarction with normal coronary arteries in Chagas' Disease may include: embolic events, particularly when there is associated congestive heart failure; coronary thrombosis and coronary spasms.

**KEY WORDS:** Chagas' disease — Myocardial infarction.

### I N T R O D U C T I O N

The occurrence of myocardial infarction with no obstructive coronary artery lesion, in chronic Chagas' Disease is not frequent.

A recent retrospective study of 1,345 autopsies of chagasic patients showed only 5 cases with embolic coronary myocardial infarction<sup>20</sup>. Other cases with anatomopathological evidence of embolic coronary event has been described, indeed, and rare cases have also been reported with obscure physiopathological mechanisms<sup>3, 14, 23, 24</sup>.

This report describes two chagasic patients with congestive heart failure, who developed acute myocardial infarction. The diagnose was based on sudden chest pain, increases of serum MB-CK level and ECG changes compatible with non-transmural MI. Coronary angiograms

performed late did not show obstructive coronary disease.

### C A S E R E P O R T S

#### Case 1

G.F.R., a 46-year-old black man had been under ambulatory treatment for chronic Chagas' cardiomyopathy with congestive heart failure for the last 10 months. He was in functional class II, taking furosemide, digoxin and potassium chloride by mouth. The diagnosis of Chagas' disease was based on classical clinical history and confirmed by complement fixation and the immunofluorescence reactions<sup>1, 7</sup>.

On February 9th, 1982 he suffered a sudden, oppressive and intense chest pain, without precipitating factor. It was followed by nausea

From the "Instituto do Coração do Hospital das Clínicas — Faculdade de Medicina da Universidade de São Paulo". São Paulo, Brasil

Address for reprints: Dra. Sílvia G. Lage — Rua Oscar Freire n.º 1549/94 — CEP 05409 — São Paulo, SP, BRASIL

and vomiting which lasted about 3 hours and lessened after intravenous meperidine. Physical examination disclosed mild dyspnea at rest. Temperature was 36.6°C. Arterial blood pressure was 120 x 80 mmHg and heart rate 108 beats/min. The jugular veins were noticeably distended. Bilateral pulmonary rales were present. A S<sub>3</sub> gallop was audible at the apical area. The liver was enlarged and tender and there was mild edema in the inferior limbs.

Routine laboratory tests were in the normal range at admission. The first serum MB-CK sampled 4 hours after the beginning of pain, was 20.5 U/l (normal value — 10 U/l); the highest value was 65 U/l, at 22 hours after the beginning of pain (Fig. 1B). Chest X-ray showed pulmonary congestion and cardiomegaly. ECG showed mild ST segment elevation (0.1 mV) in leads II, III and aVF, in the presence of pain. (Fig. 1A). There were also left ventricle hypertrophy, left anterior hemiblock and an inactive septal area.

The <sup>99m</sup>Tc-PYP myocardial scintigram was performed 72 hours after the beginning of pain, and showed increased captation of the infero-lateroapical area of the heart. (Fig. 2A).

Clinical course was uneventful until the 10th day, when a complete, proportionate left hemiplegia occurred, with total regression in 30 minutes.

A coronary angiogram performed 16 days after the myocardial infarction showed a small and smooth right coronary artery, without obstructive lesions. The left coronary artery was dominant without obstructive lesions in its ramifications (Fig. 1C). Dye velocity in the right coronary artery was somewhat slow. Left ventricular angiography displayed an increased end-systolic volume and a moderate, diffuse hypokinesis. An ECG performed at this time showed negative T waves in the inferior leads (Fig. 1A).

The patient was discharged from hospital on functional class I, on digoxin and furosemide. He returned one year later, complaining of fatigue, dyspnea and peripheral edema; he had discontinued spontaneously his medication. Five blood samples were taken for MB-CK dosage, at 4 hours interval each. They were all normal. ECG showed positive T waves in the infe-

rior leads (Fig. 1A). The <sup>99m</sup>Tc-PYP myocardial scintigram was normal (Fig. 2B). He received standard therapy for heart failure.

## Case 2

On March 1st, 1982, M.L.A.R., a 48-year-old black woman presented an intense, oppressive chest pain, irradiated to the left shoulder and arm. It was provoked by a slight physical effort, lasting about 5 hours, being lessened by intravenous meperidine. In the last year she had palpitations, atypical chest pain and dyspnea resulting from moderate efforts which lessened under rest. At admission physical examination revealed a good general health condition; the axillary temperature was 36.6°C, heart rate 75 beats/min, and the blood pressure was 140 x 60 mmHg. A S<sub>4</sub> was heard at the apical area without other abnormalities.

Routine laboratory tests were in the normal range. The complement fixation and the immunofluorescence reactions for Chagas'disease were positive<sup>17</sup>. The first serum MB-CK obtained 6 hours after the beginning of pain, was 8.6 U/l (normal value — 10 U/l) reaching its peak 77 U/l after 18 hours (Fig. 3B). Chest X-ray film displayed a mild pulmonary congestion and moderate cardiomegaly.

ECG showed sinus rhythm, right bundle branch block and left anterior hemiblock. Next day, the ECG displayed a decreased amplitude of R wave from V<sub>3</sub> to V<sub>6</sub> and T wave became negative in leads II, III, aVF and from V<sub>3</sub> to V<sub>6</sub>. Subsequently, ECG showed preservation of QRS morphology and positive waves (Fig. 3A).

Vectocardiogram confirmed an inactive area in the anterior and lateral region. A round and symmetrical T-loop in the horizontal, frontal and sagittal planes was evident suggesting an ischaemic repolarization.

Coronary angiogram was performed 9 days after the myocardial infarction. It showed and left coronary arteries without obstructive lesions (Fig. 3C). The left ventricular angiography revealed an important and diffuse hypokinesis with apical dyskinesis. The patient recovered without any other complication and

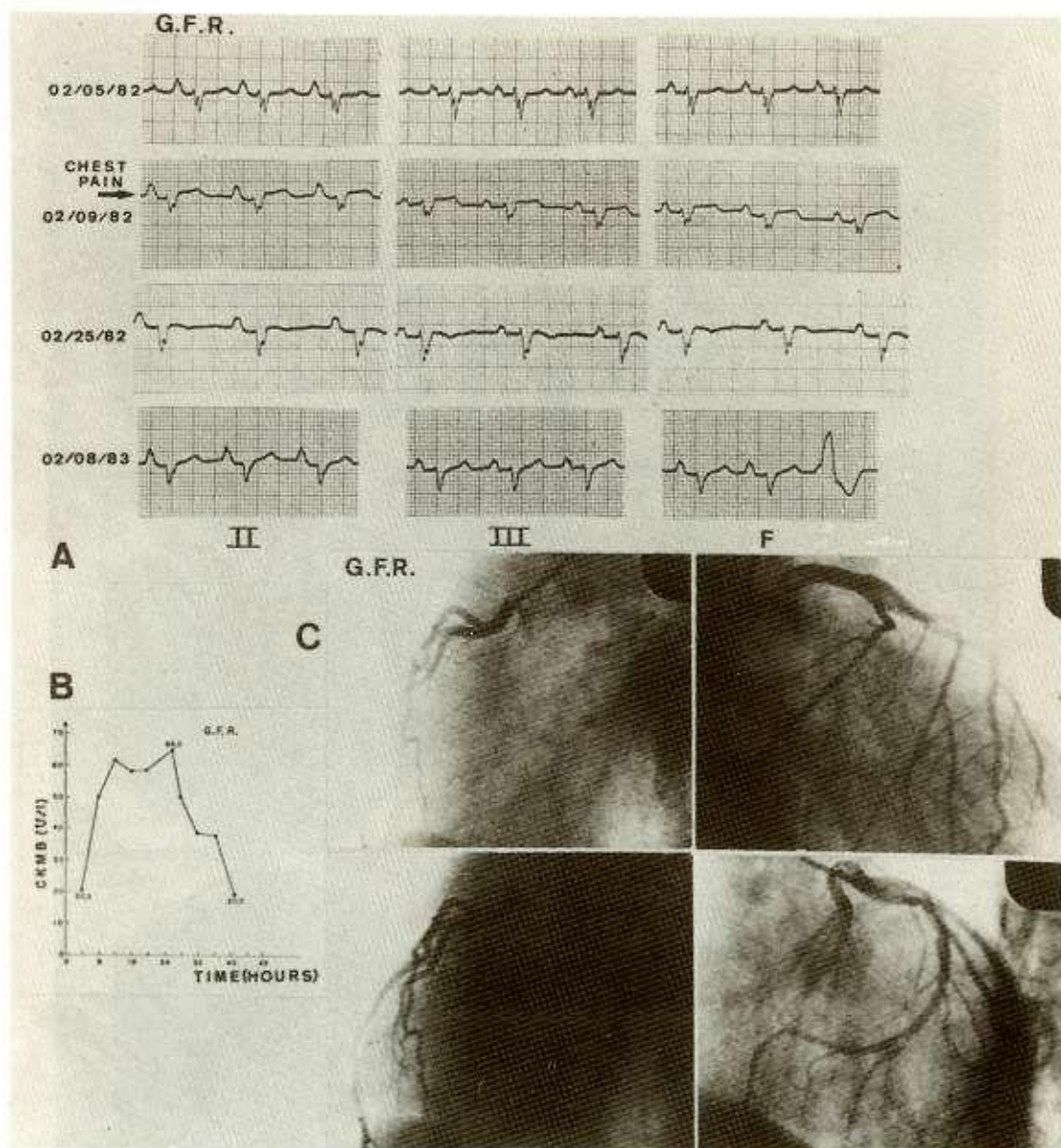


Fig. 1 — Case 1: A) — Evolutive ECG pointing out the ST segment and T wave changes at the inferior wall; B) — CK-MB curve showing the progressive increase of enzyme values; peak (65 U/l) is reached after 22 hours of the chest pain; C) — Right and left coronary arteriogram without obstructive lesions.

was discharged from hospital 15 days after being admitted.

#### COMMENTS

The cases here in reported are typical examples of patients with congestive heart failure due to Chagas' disease. Their unique feature

is that they presented clinical, eletrocardiographic and enzymatic alterations of acute myocardial infarction (AMI), in the presence of normal coronary arteries as documented by angiography. The incidence of AMI associated with coronaries without obstructive lesions varies from 1 to 12%<sup>6,10</sup>. Such variation depends on the methodology used for the diagnosis (ana-

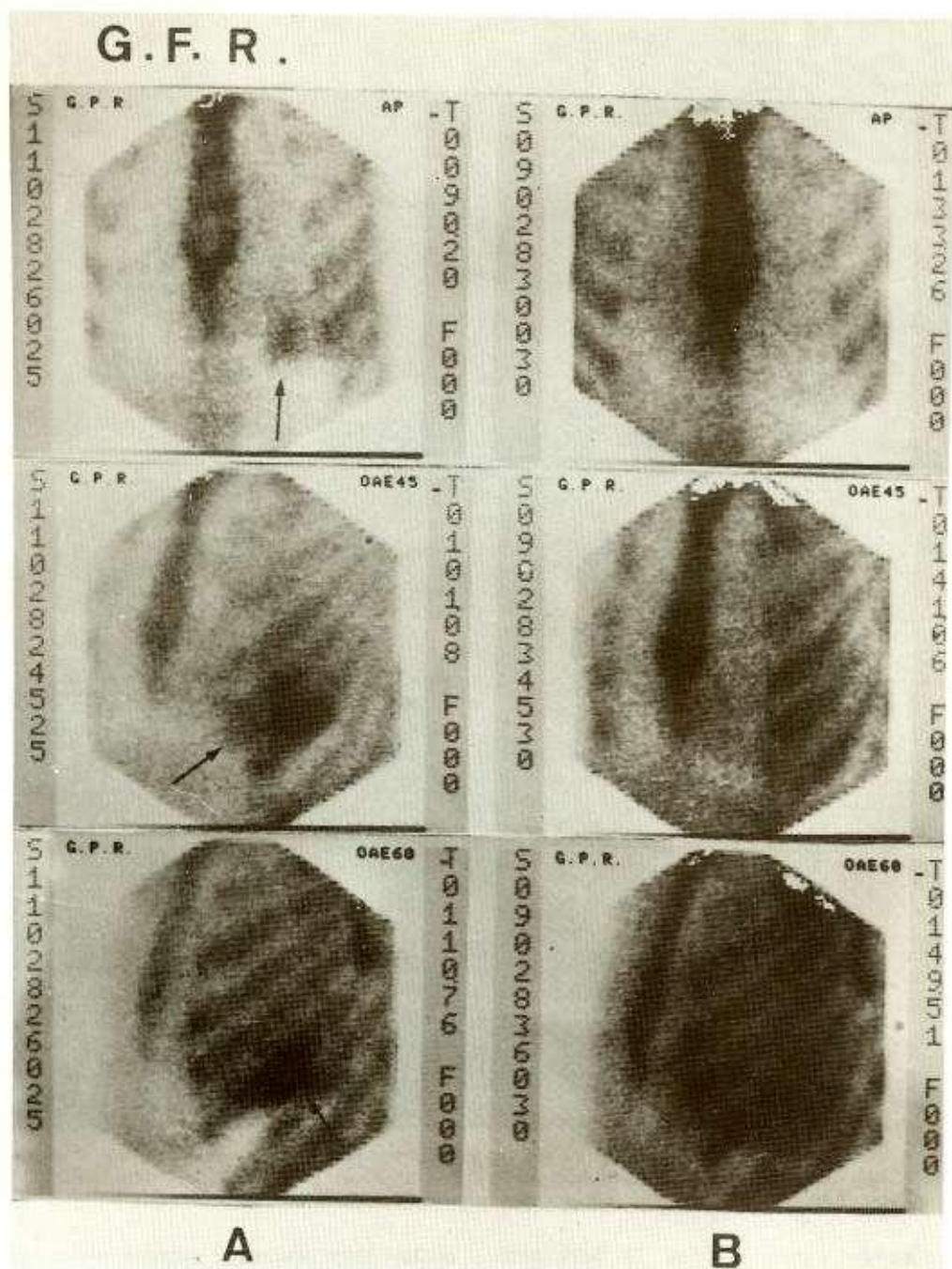


Fig. 2 — Case 1: A) —  $^{99m}\text{Tc}$ -PYP myocardial scintigram performed 72 hours after the beginning of the pain; abnormal areas at the inferior, lateral and apical regions are seen (arrows); B) — Normal  $^{99m}\text{Tc}$  PYP myocardial scintigram performed one year later.

tomopathologic or angiographic method) and on the time elapsed between the acute and the angiographic investigation.

Presumably the basic phenomenon is a temporary interruption of the coronary flow, that is caused by embolism, thrombosis or coronary

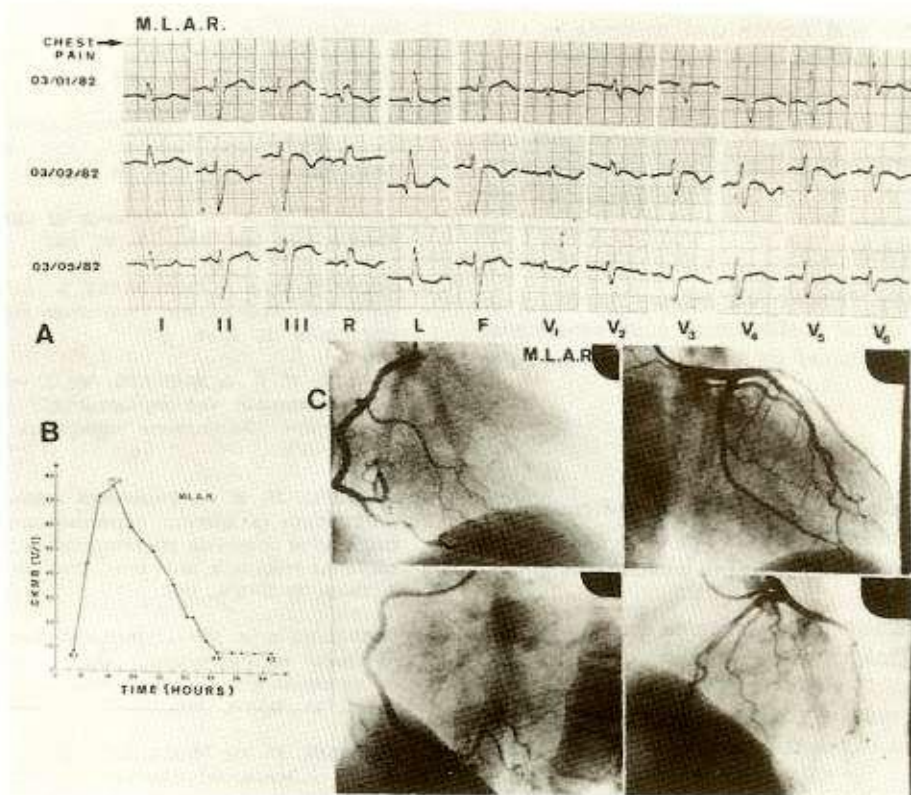


Fig. 3 — Case 2: A) — Evolutive ECG pointing out the ST segment and the T wave changes at the inferior and anterior walls; B) — CK-MB curve showing the progressive increase of enzyme values; peak (77 U/l) is reached after 18 hours of the chest pain; C) — Right and left coronary arteriogram without obstructive lesions.

spasm<sup>9,15,21</sup>. These factors can operate either separately or together, with a subsequent thrombolysis or vessel wall dilatation, that could explain the transitory cessation of coronary flow in the presence of a normal coronary or of minimal lesions of the coronary endothelium, no matter how imperceptible they can be to the coronary angiogram<sup>12,13,18,19</sup>.

In Chagas' disease there are peculiar anatomical and functional factors that may suggest that AMI with monobstructive coronary artery lesions represents more than a casual association. One is the presence of minimal coronary endothelial lesions that have been experimentally reported<sup>16,17</sup>. Although these alterations have never been reported in human, their presence cannot be excluded as a predisposing factor. Another is the typical parasympathetic denervation of Chagas' disease that could play a role in the neuronal imbalance of the coro-

anry tonus<sup>7,4,11</sup>. This could facilitate the development of coronary spasms.

Coronary embolism with subsequent spontaneous resolution is another potential mechanism of infarction. It is known that chronic chagasic cardiomyopathy is a congestive stage is a potentially embolic disease<sup>5,8,20,22</sup>. The organs more frequently affected by embolism are lungs, kidneys, spleen, brain and more rarely the heart. The embolic source is often the right atrium or the left ventricular apex<sup>2,3,20</sup>. Therefore, coronary embolism is a rare but acceptable hypothesis to explain these cases.

We conclude that it is not possible to indicate with certainty any physiopathological mechanisms responsible for the myocardial infarction in the cases reported above. The evidence of predisposing factors for the thrombus formation and the possibility of coronary spasm, make us suppose that the occurrence

of ischaemia and myocardial necrosis in Chagas' disease, despite rare, can represent more than a coincidental nosological association.

Although clinical recognition of AMI is scarce among population where Chagas' disease has a high prevalence, this may be due to lack of identification the atypical AMI, than to its rare occurrence.

Naturally, further studies are necessary to assess the actual incidence of such association and its importance to the future therapeutic decisions.

### RESUMO

#### Infarto agudo do miocárdio na cardiomiopatia chagásica crônica. Relato de dois casos com coronárias sem lesões obstrutivas

São relatados dois pacientes com doença de Chagas, forma cardíaca crônica, que desenvolveram Infarto Agudo do Miocárdio (IAM). Ambos apresentaram dor precordial súbita em opressão, sem fatores precipitantes. No primeiro o pico de CKMB foi 65 U após 22 horas do início da dor e no segundo foi de 77U após 18 horas. O ECG em ambos evidenciou apenas alterações sugerindo IAM não transmural.

A cintilografia miocárdica com  $^{99m}\text{Tc}$ -PYP foi positiva no primeiro caso.

1 coronariografia realizada respectivamente no 16.º e 9.º dia não evidenciou lesões obstrutivas.

São discutidos os possíveis mecanismos de IAM com coronárias sem lesões obstrutivas na doença de Chagas tais como: eventos embolíticos, trombóticos e espásticos.

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### REFERENCES

1. ALMEIDA, J. O. & FIFE, J. E. F. — Métodos de fijación del complemento estandarizado cuantitativamente para la evaluación crítica de antígenos preparados con *Trypanosoma cruzi*. Washington, Organización Panamericana de Saúde, 1976. (Publicação Científica no. 319).

2. ANDRADE, Z. A. & ANDRADE, S. G. — A patologia da doença de Chagas (Forma crônica cardíaca). *Bol. Fund. G. Moniz*, 6: 1-53, 1955.
3. ANDRADE, Z. A. — Fenômenos tromboembólicos na cardiopatia crônica chagásica. *Anais do Congresso Internacional sobre Doença de Chagas*, 1: 73-84, 1959.
4. ANDRADE, Z. A. — Pathogenesis of Chagas' disease. *Braz. J. med. biol. Res.*, 15: 187, 1982.
5. ANDRADE, Z. A. & SADIGURSKY, M. — Tromboembolismo em chagásicos sem insuficiência cardíaca. *Gaz. méd. Bahia*, 71: 59-64, 1971.
6. ARNETT, E. N. & ROBERTS, W. C. — Acute myocardial infarction and angiographically normal coronary arteries. An unproven combination. *Circulation*, 53: 395, 1976.
7. CAMARGO, M. E. — Fluorescent antibody teste for the diagnosis of American Trypanosomiasis. Technical modification employing preserved culture forms of *Trypanosoma cruzi* in a slide test. *Rev. Inst. Med. trop. S. Paulo*, 8: 227-234, 1966.
8. CARVALHO, J. A. M. — Tromboembolismo na doença de Chagas em Pernambuco. Considerações em torno da incidência em material necroscópico. *Rev. bras. malar.*, 15: 611-616, 1963.
9. CHEITLIN, M. D.; McALLISTER, H. A. & CASTRO, C. M. — Myocardial infarction without atherosclerosis. *J. Amer. med. Ass.*, 231: 951-959, 1975.
10. KHAN, A. H. & HAYWOOD, L. J. — Myocardial infarction in nine patients with radiologically patent coronary arteries. *New Engl. J. med.*, 291: 427, 1974.
11. KOBERLE, F. — Cardiopathia parasymphaticopriva. *Münch. med. Wschr.*, 101: 1308, 1959.
12. MASERI, A.; L'ABBATE, A.; BAROLDI, G.; CHIERCHIA, S.; MARZILLE, M.; BALESTRA, A. M. et al. — Coronary vasospasm as a possible cause of myocardial infarction. *New Engl. J. Med.*, 299: 1271-77, 1978.
13. MASERI, A. & CHIERCHIA, S. — Coronary artery spasm: demonstration, definition, diagnosis and consequences. *Prog. cardiovasc. Dis.*, 25: 169-192, 1982.
14. MONTES PAREJA, J.; AMARGOS, A.; ESTABLE, J. J. & FERREIRA-BERRUTTI, P. — Forma cardíaca de la Tripanosomiasis cruzi. *Arch. urug. Cardiol.*, (apartado) 1-15, 1938.
15. NEMERSON, Y. & NOSSEL, H. L. — The biology of thrombosis. *Ann. Rev. Med.*, 33: 479-88, 1982.
16. OKUMURA, M.; CORRÊA NETO, A. & SILVA, A. C. — Contribuição para o estudo da patogenia das lesões vasculares na doença de Chagas experimental em camundongos brancos. *Rev. paul. Med.*, 61: 265-266, 1962.
17. OKUMURA, M.; BRITO, T.; SILVA, L. H. P. & CORRÊA NETO, A. — The pathology of experimental Cha

LAGE, S. G.; MANSUR, A. P.; RAMIRES, J. A. F.; LUZ, P. da; BELLOTTI, G. & PILEGGI, F. — Acute myocardial infarction in chronic Chagas'cardiomyopathy. Report of two cases with no obstructive coronary artery lesions. *Rev. Inst. Med. trop. São Paulo*, 28:131-137, 1986.

- ga's disease in mice. I. Digestive tract changes with a reference to necrotizing arteritis. *Rev. Inst. Med. trop. S. Paulo*, 2: 17-28, 1960.
18. OLIVA, P. B. & BRINCKINRIDGE, J. C. — Arteriographic evidence of coronary arterial spasm in acute myocardial infarction. *Circulation*, 56: 366-374, 1977.
19. OLIVA, P. B. — Pathophysiology of acute myocardial infarction. *Ann. Intern. Med.*, 94: 236-250, 1981.
20. OLIVEIRA, J. S. M.; CORREA DE ARAUJO, R. R.; NAVARRO, M. A. & MUCCILLO, G. — Cardiac thrombosis and thromboembolism in chronic Chagas'heart disease. *Amer. J. Cardiol.*, 52: 147-151, 1983.
21. RAIZNER, A. E. & CHAHINE, R. A. — Myocardial infarction with normal coronary arteries. In: HURST, J. W., ed. — *The Heart: Update I*. New York, McGraw-Hill, 1979. p. 147-166.
22. ROMANO, J. R.; SILVA, A. Q.; CUNHA, G. P. & OLIVEIRA, P. F. — Tromboembolismo na doença de Chagas. *Rev. méd. Paraná*, 25: 263-276, 1956.
23. VIANNA, G. L.; CAMPOS, G. P. & MAGALHÃES, A. V. — Infarto do miocárdio sem obstrução coronariana associado a cardiopatia chagásica crônica. *Arch. bras. Cardiol.*, 33: 41-47, 1979.
24. WAINRICH, A.; FABIUS, A.; CIRILE, J. P. & ACOSTA-FERREIRA, W. — Infarto do miocárdio en la cardiopatia chagásica. *Tórax*, 4: 255, 1955.

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