The Impact of Syncope During Clinical Presentation of Sustained Ventricular Tachycardia on Total and Cardiac Mortality in Patients with Chronic Chagasic Heart Disease

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Objective - *To assess the impact of syncope during sustained ventricular tachycardia on total and cardiac mortality in patients with chronic chagasic heart disease.*

Methods - *We assessed 78 patients with sustained ventricular tachycardia and chronic Chagas' heart disease. The mean age was 53±10 years, 45 were males, and the mean ejection fraction was 49.6±13%. The patients were divided into 2 groups according to the presence (GI=45) or absence (GII=33) of syncope during sustained ventricular tachycardia.*

Results - *After a mean follow-up of 49 months, total mortality was 35% (28 deaths), 22 deaths having a cardiac cause (78.6%). No difference was observed in total (33.3% x 39.4%) and cardiac (26.7% x 30.3%) mortality, or in nonfatal sustained ventricular tachycardia between GI and GII patients (57.6% x 54.4%, respectively). However, the presence of syncope during recurrences was significantly greater in those patients who had had the symptom from the beginning (65.4% x 18.1%, p<0.01).*

Conclusion - *Syncope during the presentation of sustained ventricular tachycardia is not associated with an increase in total or cardiac mortality in patients with chronic Chagas' heart disease. However, syncope during the recurrence ventricular tachycardia is greater in patients experiencing syncope in the first episode, of sustained ventricular tachycardia.*

Keywords: syncope, sustained ventricular tachycardia, chronic Chagas' heart disease

Identification of patients at high risk of cardiac death is fundamental in cardiological investigation. The importance of syncope during the clinical presentation of sustained ventricular tachycardia, as a prognostic factor, was extensively studied in patients with coronary artery disease, and it was considered a worse prognosis when compared with hemodynamically well-tolerated sustained tachyarrhythmias [1-5]. In chronic chagasic heart disease, however, the relevance of syncope during clinical presentation of sustained ventricular tachycardia is not totally understood, and its incidence is variable according to reports published in the literature [6-10]. The objective of this study was to assess the impact of syncope during the clinical presentation of sustained ventricular tachycardia on total, cardiac, and sudden death in patients with chronic chagasic heart disease.

Methods

We consecutively assessed 78 patients with chronic Chagas' heart disease after an episode of sustained ventricular tachycardia in the clinical electrophysiology section of the Hospital São Paulo of the Universidade Federal de São Paulo. Forty-five patients were males and 33 were females, their mean age being 53±10 years (29 to 74). The mean left ventricular ejection fraction was 49.6% ± 13% (24 to 76), and 24 (30.8%) patients had ejection fraction < 40%. In this population, 11.5% of the patients had advanced heart failure (NYHA functional class III or IV).

All patients had their diagnosis of Chagas' disease confirmed by serological tests, and the presence of heart disease was confirmed by alterations on the 12-lead electrocardiogram, chest radiography, ambulatory electrocardiographic monitoring (Holter), echocardiogram, radionuclide ventriculography, and left ventricular angiography. Monomorphic spontaneous sustained ventricular tachycardia was recorded on an electrocardiogram or on Holter monitoring in all patients. Coronary angiography was performed in all males older than 35 years and in all females older than 40 years.

The electrophysiological study with programmed ventricular stimulation was performed in all patients, according to stimulation techniques previously reported [10-13], using up to 3 extrastimuli with a minimum coupling of 200ms, under 2 cycles of basal stimulation (600 and 450ms) [10-13].

The patients were classified according to the presence (group I) or absence (group II) of syncope during clinical presentation of sustained ventricular tachycardia.

To comply with the objectives of the study, the following definitions were used: 1) syncope, the sudden loss of consciousness followed by spontaneous recovery, considered present when the episode occurred during electrocardiographic monitoring or when, after regaining consciousness, the patient was in sustained ventricular tachycardia; 2) sustained ventricular tachycardia, which is the presence of consecutive ventricular beats with a heart rate >100bpm and duration >30s, or the need for immediate reversion, when hemodynamic impairment occurs; 3) sudden cardiac death, in the case of a witnessed death, occurring within 1 hour at most after symptom onset in a clinically stable patient, or in the case of an unwitnessed death, the patient should have been asymptomatic within the 24 hours preceding death; 4) cardiac death, the sum of sudden death and that resulting from progression of heart failure or thromboembolic events; 5) recurrence of nonfatal sustained ventricular tachycardia, recorded on an electrocardiogram or Holter, with no evolution to ventricular fibrillation or cardiopulmonary arrest.

After hospital discharge, the patients were assessed monthly during the first 6 months, and then bimonthly during the first year. During follow-up, the following events were defined: death due to cardiac causes (sudden and nonsudden), to noncardiac causes, deaths after a maximum follow-up of 10 years, and recurrence of nonfatal sustained ventricular tachycardia. The initial treatment was considered the one used since hospital discharge, and maintenance treatment comprised the drugs used since the last contact.

Continuous variables were presented as mean ± standard deviation. For comparison between the groups, the chi-square test or Fisher exact test, when appropriate, was used for qualitative variables, and Student t test was used for quantitative variables. Survival curves were depicted according to the Kaplan-Meier method and compared by the log-rank test.

Results

Of the 78 patients, 45 (57.7%) experienced syncope during clinical presentation of sustained ventricular tachycardia and comprised group I; the 33 (42.3%) patients who did not experience syncope comprised group II. Other symptoms present during sustained ventricular tachycardia were palpitation in 67 patients (GI=39 and GII=28, p=0.50) and dyspnea in 38 patients (GI=21 and GII=17, p=0.40). The clinical characteristics of both groups are shown in table I. No difference between group I and II patients was observed in regard to age (52.5±10 and 53.2±10, p=0.31), sex, presence of advanced heart failure, left ventricular ejection fraction (48.1±13 and 51.6±13, p=0.8), number

of patients with ejection fraction <40% [GI=16 (35.6%) and GII=8 (24.2%), (p=0.21)], and segmentary alteration (77.8% and 69.7%, p=0.29). Density of isolated ventricular extrasystoles per hour (193 vs 91, p=0.04) and nonsustained ventricular tachycardia in 24 hours (70 vs 8, p=0.25) recorded on the initial Holter was greater in the patients with syncope.

Most patients had electrocardiographic alterations, which were present in 80% (n=36) of the patients with syncope and 78.8% (n=26) of those without. The most common electrocardiographic findings were as follows: right bundle-branch block associated with left anterosuperior divisional block (29.5%), isolated right bundle-branch block (10.3%), isolated left bundle-branch block (10.3%), and left divisional anterosuperior block (11.5%). None of these alterations predominated in the patients experiencing or not experiencing syncope.

The frequency cycle of clinical sustained ventricular tachycardia was analyzed in 49 patients (GI=25, GII=24). The mean frequency cycle in GI was 314 ms [95% CI 282-347], and, in GII, it was 309 ms [95% CI 282-337], with no statistically significant difference between the groups (p=0.79).

During electrophysiological study with programmed ventricular stimulation, sustained ventricular tachycardia was induced in 75 patients. The frequency cycle of induced sustained ventricular tachycardia was 296 ms [95% CI 273-319] in the patients experiencing syncope and 325 ms [95% CI 299-351] in those not experiencing syncope (tab. I). A tendency toward a shorter frequency cycle of induced sustained ventricular tachycardia was observed in the patients experiencing syncope (p=0.08).

All chagasic patients with sustained ventricular tachycardia were successfully treated and were available for ambulatory follow-up. The cardiac drugs used during the last contact are listed in table II. At the end of follow-up, 71 patients were using amiodarone, 41 (91.1%) in the group of

Table I – Baseline characteristics of patients with sustained ventricular tachycardia and chronic chagasic heart disease according to the presence of syncope during clinical presentation of the arrhythmia			
	Group I with syncope (N=45)	Group II without syncope (N=33)	P
Age (years)	52.5±10	53.2±10	0.31
Male sex (%)	29 (54.4)	16 (48.5)	0.12
Abnormal ECG (%)	36 (80)	26 (78.8)	0.91
Segmentary alteration (%)	35 (77.8)	23 (69.7)	0.29
Ejection fraction	48.0 ±13	51.6±13	0.79
Ejection fraction <40% (%)	16 (35.6)	8 (24.2)	0.21
NYHA Functional class III and IV (%)	13.3%	9.1%	0.41
Frequency cycle of clinical SVT (ms)	314±78	309±65	0.79
Frequency cycle of induced SVT (ms)	296±78	325±71	0.08
Isolated VE/h	193	91	0.04*
NSVT/24h	70	8	0.25
Follow-up duration (months)	47±27	42±25	0.80

ECG- electrocardiogram; SVT- sustained ventricular tachycardia; VE-ventricular extrasystoles; NSVT- nonsustained ventricular tachycardia.

patients experiencing syncope and 30 (90.9%) in those not experiencing syncope (p=0.21). Six patients were using sotalol, and 2 patients were not using antiarrhythmic drugs. Catheter ablation was performed in 34 patients, 18 (40%) patients in group I and 16 (45%) patients in group II (p=0.50). The use of a definitive pacemaker for bradyarrhythmias did not differ in the 2 groups. Six (13.3%) patients experiencing syncope and 7 (21.2%) not experiencing syncope used pacemakers implanted for bradyarrhythmias.

The mean follow-up time (fig. 1) was 49±33 months, and all patients were followed up for at least 1 year, except 1 patient in GI who was followed up for 10 months. In group I, the mean follow-up was 51±32 months, and 7 (15.5%) patients in this group were followed up for less than 24 months;

in group II, the mean follow-up was 46±32 months, and 5 (15.1%) patients were followed up for less than 24 months.

Total mortality was 35% (28 deaths). No difference between the groups was observed in regard to total mortality; 15 (33.3%) patients died in group I, and 13 (39.4%) patients died in group II (tab. III). The survival curve showed that, during the entire clinical follow-up, total mortality was similar in both groups (fig. 2).

Of the deaths in the 78 patients, 28.2% had cardiac causes, accounting for 78.6% of deaths in this population (22 deaths). Figure 3 shows the curve of accumulated cardiac deaths in the 78 patients, which did not statistically differ between groups I and II (p=0.64), 12 (26.7%) and 10 (30.3%) being the deaths from cardiac causes, respectively. Most cardiac deaths occurred suddenly. Of all cardiac deaths, 63.6% were considered sudden (14 deaths). Sudden death accounted for 50% (6/12) of the cardiac deaths in group I patients and for 80% (8/10) of the deaths in group II patients (p=0.24). Mean survival in group I was 83 months [95% CI 70-97], and in group II it was 75 months [95% CI 59-92] (tab. III).

Recurrence of nonfatal sustained ventricular tachycardia was observed in 44 (56.4%) patients, 26 being in group I (57.8%) and 18 in group II (54.5%). No difference between the 2 groups was observed in the recurrence of nonfatal sustained ventricular tachycardia (fig. 4). Recurrences of sustained ventricular tachycardia were accompanied by syncope in 26.9% of the patients, being more frequent in those who already had the symptom prior to antiarrhythmic

Table II – Pharmacological and antiarrhythmic treatment			
	Group I with syncope N=(45)	Group II without syncope N=(33)	P
Digitalis	10 (22.2%)	7 (21.2%)	1.00
ACE I	28 (62.2%)	24 (72.7%)	0.46
Diuretics	15 (33.3%)	17 (51.5%)	0.16
Amiodarone	41 (91.1%)	390.2±73.5	0.21
Dose média (mg)	30 (90.9%)	386.6±68.1	0.83
Sotalol	5 (15.1%)	1 (3%)	0.31
mean dose (mg)	224±35	160	
Catheter ablation	18 (40%)	16 (45%)	0.50
Definitive pacemaker	6 (13.3%)	7 (21.2%)	0.75

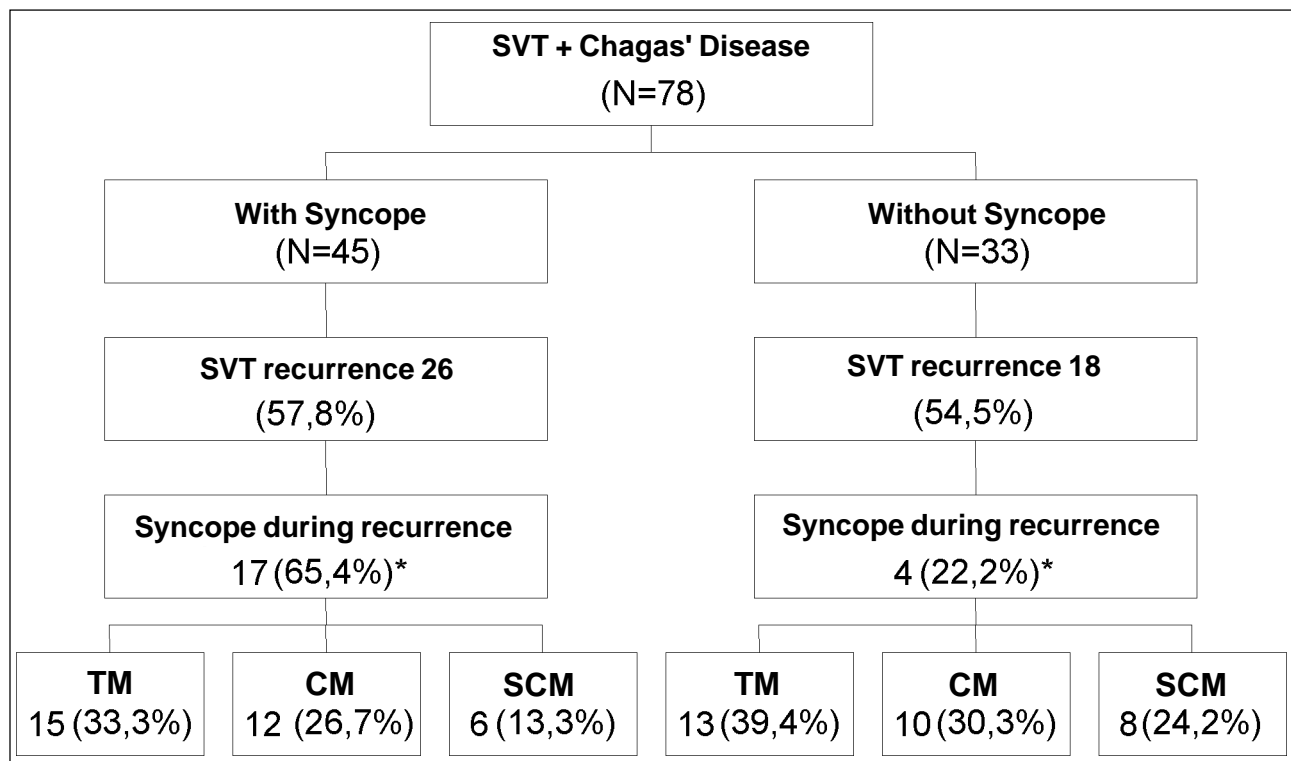ACE I - angiotensin-converting enzyme inhibitors.

Fig. 1 – Events during clinical follow-up of patients with chronic chagasic heart disease according to the presence of syncope during clinical presentation of sustained ventricular tachycardia. TM- total mortality; CM- cardiac mortality; SCM- sudden cardiac mortality.

Table III – Total, cardiac, and sudden mortality, and recurrence of nonfatal sustained ventricular tachycardia in patients with chronic chagasic heart disease

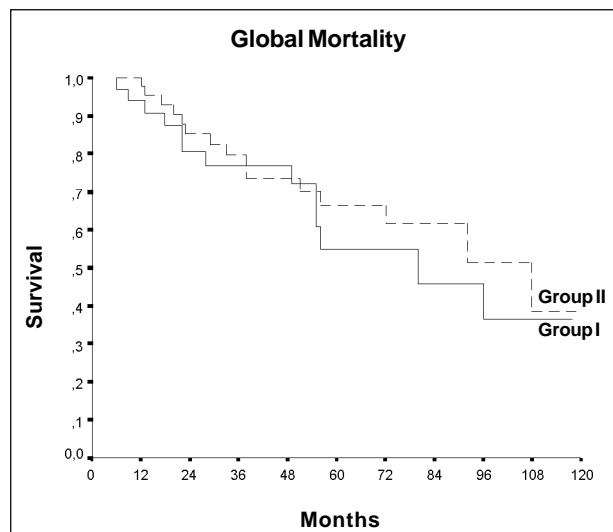|	Total Mortality (N=28)	Cardiac mortality (N=22)	Sudden mortality (N=14)	SVT recurrence	Survival (m) [95% CI]	Recurrence-free time (m) [95% CI]
Group I – with syncope (N=45)	15 (33.3%)	12 (26.7%)	6 (50%)	26 (57.8%)	83 [70; 97]	71 [59; 83]
Group II – without syncope (N=33)	13 (39.4%)	10 (30.3%)	8 (80%)	18 (54.5%)	75 [59; 92]	64 [50; 78]
P	0.43	0.59	0.24	0.82	0.50	0.59

Fig. 2 – Total survival curve of patients with chronic chagasic heart disease and sustained ventricular tachycardia according to the presence of syncope during clinical presentation of the arrhythmia. Group I- with syncope; group II- without syncope.
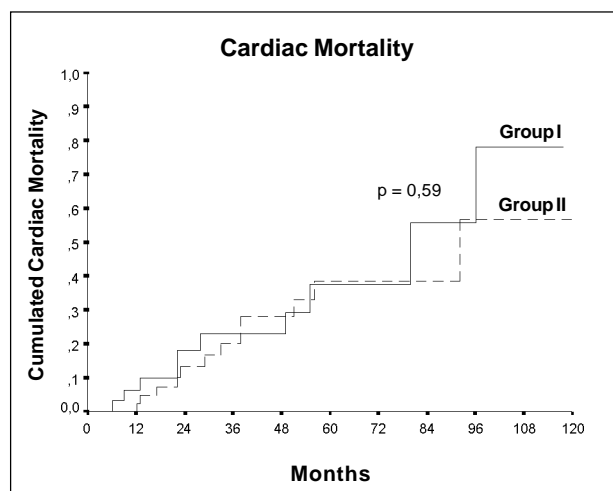

Fig. 3 – Curve of accumulated cardiac mortality in patients with chronic chagasic heart disease and sustained ventricular tachycardia according to the presence of syncope during clinical presentation of the arrhythmia. Group I- with syncope; group II- without syncope.

therapy. In the 26 group I patients with recurrence of nonfatal sustained ventricular tachycardia, 17 (65.4%) experienced syncope; in the 18 group II patients with recurrence

of nonfatal sustained ventricular tachycardia, 4 (22.2%) experienced syncope (p<0.01).

To assess the importance of syncope associated with sustained ventricular tachycardia, the patients experiencing syncope at any time during the study were compared with those not experiencing it. Therefore, syncope associated with sustained ventricular tachycardia occurred in 4 of the 33 patients whose arrhythmia had initially occurred without syncope, adding to the 49 patients who experienced syncope associated with sustained ventricular tachycardia at any time during the study. When these 49 patients with syncope associated with sustained ventricular tachycardia were compared with the 29 without syncope, total (36.7% vs. 34.5%, p=0.91) and cardiac (24.5% and 34.5%) mortality did not differ at any time during the study.

Total and cardiac mortality was greater in the patients with left ventricular ejection fraction lower than 40%. Of the 28 deaths occurring during total clinical follow-up, 50% of the patients had an ejection fraction lower than 40% as compared with 30.8% of the total population and 29% of the patients without this event; this difference, however, was not statistically significant.

Discussion

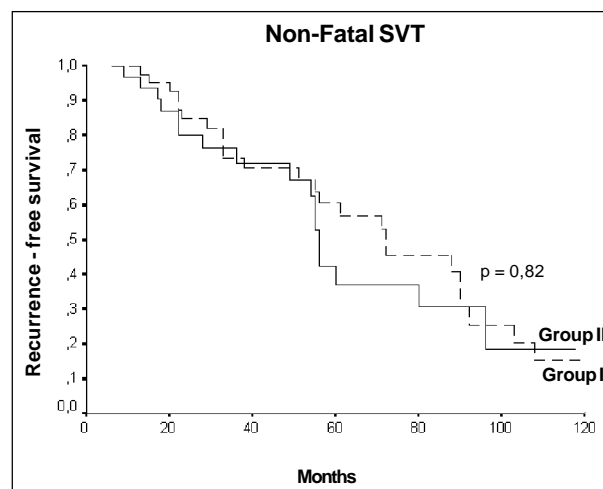Our study assessed the impact of syncope during the

Fig. 4 – Recurrence-free survival of nonfatal sustained ventricular tachycardia in patients with chronic chagasic heart disease and sustained ventricular tachycardia according to the presence of syncope during clinical presentation of the arrhythmia.

clinical presentation of sustained ventricular tachycardia on total and cardiac mortality in patients with chronic chagasic heart disease. In our case series, the presence of syncope during sustained ventricular tachycardia, even though frequent (57.7%), did not influence total and cardiac mortality in the long run.

Sustained ventricular tachycardia in patients with structural heart disease is a potentially lethal arrhythmia. Previous studies reported that, in the presence of structural cardiac impairment, mainly in ischemic heart disease, when sustained ventricular tachycardia is accompanied by syncope or cardiopulmonary arrest, the risk of a fatal recurrence is greater than when this arrhythmia is hemodynamically well tolerated [1-5]. In chronic chagasic heart disease, however, the impact of the presence of syncope during clinical presentation of sustained ventricular tachycardia has been studied less [6-14].

Scanavacca et al [9], studied 35 patients with sustained ventricular tachycardia and chronic chagasic heart disease treated with amiodarone. They showed that, despite recurrence of arrhythmia in 30% of the patients, no death was observed in patients with an ejection fraction >30% and who were in functional class I or II during 27 months of clinical follow-up. In their study, cardiac mortality was 11.4%, occurring in patients with severe ventricular dysfunction. However, the authors did not evaluate mortality according to the clinical presentation of the arrhythmia. Mendoza et al [15] reported a mortality rate of 20% in patients with sustained ventricular tachycardia and chronic chagasic heart disease; however the importance of clinical presentation during the spontaneous arrhythmia in the evolution of these patients was not mentioned. In our study, total (33.3% vs. 39.4%, p=0.43) and cardiac (26.7% vs. 30.3%, p=0.59) mortality was similar in patients with and without syncope. These results may not be attributed to differences in clinical, electrophysiological, or therapeutical characteristics, because they were similar for both groups, and most patients were receiving amiodarone. The higher mortality rate in our study as compared with that in other studies may have been due to the larger size of our sample and our longer follow-up.

It is important to emphasize that when we analyzed only the patients with ejection fraction below 40%, total, cardiac, and sudden deaths were similar in the patients with and without syncope. However, contrary to that which happens in coronary artery disease, but similar to that reported in other studies involving only patients with Chagas' disease [6,15-18], sustained ventricular tachycardia occurred in patients with mild ventricular dysfunction, as shown by the mean ejection fraction of 49.6%, and in only 30.8% of the patients with ejection fraction below 40%. Bestetti et al [6] reported ventricular dysfunction in 50% of the chagasic patients with sustained ventricular tachycardia studied with echocardiography; only 10% had severe dysfunction. In patients undergoing catheter ablation, Sosa et al [16] reported a mean ejection fraction of 62%, measured on echocardiography; these results were similar to those reported by Mendoza et al [15], studying 15 chagasic patients with sustained ventricular

tachycardia (mean ejection fraction of 56%). The small number of patients with severe ventricular dysfunction in our population was certainly a limitation in evaluating the importance of syncope associated with sustained ventricular tachycardia when the ejection fraction was reduced.

In our study, we could also observe a similar recurrence rate of sustained ventricular tachycardia in patients with and without syncope (57.8% vs. 54.5%, p=0.82); syncopal sustained ventricular tachycardia, however, was more frequent in patients who had the symptom from the beginning [17/26 (65.4%) vs. 4/18 (22%), p<0.01]. Our data suggest that the presence of syncope during sustained ventricular tachycardia does not increase the probability of recurrence. Patients with previous episodes of syncope, however, more commonly experience this symptom during recurrences of sustained ventricular tachycardia. Likewise, Scanavacca et al [9] reported a 56% probability of recurrence of sustained ventricular tachycardia in 36 months, which was similar for patients with and without syncope or resuscitated sudden death at the beginning of the study. No reference to the presence or absence of syncope associated with recurrences was made in their study results.

Syncope in chronic chagasic heart disease may result from 3 factors: tachyarrhythmias, bradyarrhythmias, and autonomic dysfunction. Syncope associated with sustained ventricular tachycardia has a variable incidence reported in the literature [6-10]. In the study by Bestetti et al [6], 3 out of 15 chagasic patients with sustained ventricular tachycardia had syncope, but only 1 during arrhythmia. Scanavacca et al [9], in a similar population, reported syncope in 37% of the 35 patients studied. In patients with sustained ventricular tachycardia and chronic chagasic heart disease, who had undergone epicardial ablation, Sosa et al [16] reported a 60% incidence of syncope and presyncope. This result was similar to the 62.5% incidence reported by de Paola et al [10] in chagasic patients with sustained ventricular tachycardia undergoing angiographic and electrophysiological studies, and to the 58% found in our study.

Even though syncope has been studied in the context of chronic chagasic heart disease, specifically during the clinical presentation of sustained ventricular tachycardia, its importance as a prognostic determinant is not totally known. Martinelli et al [13] carried out an electrophysiological study in 53 chagasic patients with recurring syncope and reported that mortality was significantly greater in the patients who had induced sustained ventricular tachycardia during programmed ventricular stimulation. Mendonça et al [14] showed that inducibility of sustained ventricular tachycardia was greater in patients with nonsustained ventricular tachycardia and syncope, who evolved with cardiac death (45.4% x 14.2%, p<0.05). In these studies, however, no reference was made to the presence of syncope associated with spontaneous sustained ventricular tachycardia. Bestetti et al [6], studying 74 patients with chagasic heart disease, reported that syncope was not a predictor of sudden death in this population. The authors did not include exclusively patients with sustained ventricular tachycardia, and, there-

fore, the importance of the symptom when associated with the arrhythmia could not be determined.

The hemodynamic response to sustained ventricular tachycardia depends on several factors, which include ventricular dysfunction, atrioventricular synchronism, the frequency cycle of ventricular tachycardia, and the response of the autonomous nervous system [19-20]. However, the response of blood pressure and severity of the symptoms may vary even in patients with similar ventricular function and ventricular tachycardia frequency cycles [19-23].

The frequency cycle of sustained ventricular tachycardia has been valued as 1 of the major markers of the hemodynamic response to ventricular tachycardia. Adhar et al [21] reported that the frequency cycle of ventricular tachycardia induced by electrophysiological study was shorter when clinical ventricular tachycardia manifested with syncope. However, this difference did not persist after multivariate analysis. On the other hand, Landolina et al [22] showed that hemodynamic deterioration resulted from impaired baroreflex sensitivity, and not from the ejection fraction and the frequency cycle of tachycardia. In our study, the frequency cycle of clinical sustained ventricular tachycardia of the patients with syncope (314 ms, 95% CI 282-347) did not significantly differ from that observed in patients without syncope (309 ms, 95% CI 282-337). However, a tendency (0.08) towards a shorter frequency cycle of induced sustained ventricular tachycardia was observed in patients in group I [296 ms (95% CI 273-319)] as compared with those in group II [325 ms (95% CI 299-351)]. Therefore, the presence of syncope in group I patients cannot be explained by the difference in the frequency cycle of sustained ventricular tachycardia.

Recently, Landolina et al [22] and Hamdan et al [23] reported that impaired baroreflex sensitivity correlated with hemodynamic deterioration during sustained ventricular tachycardia, and that the gain in arterial baroreflex significantly contributed to hemodynamic stability after initial deterioration. These studies were carried out in patients with coronary artery disease, in which severe impairment of the autonomous nervous system is an important prognostic factor, because it denotes an advanced stage of heart failure, the major predictor of mortality. Chagas' disease may affect the autonomous nervous system, independently from advanced cardiac impairment, or without advanced heart failure, and this may partially explain the mechanism of syncope in these patients, without resulting in an increase in mortality. However, because in our study no assessment of the autonomous nervous system was performed, no conclusion can be drawn in regard to its role in the occurrence of syncope in this population.

In conclusion, our data suggest that the presence of syncope during sustained ventricular tachycardia did not influence total, cardiac, or sudden mortality in the chagasic population treated with amiodarone, despite the high recurrence rate of the arrhythmia. In addition, the probability of occurrence of syncope during the recurrence of sustained ventricular tachycardia was greater in the patients who had previously experienced the symptom. Until more specific predictors of risk are identified, the management of chagasic patients who have sustained ventricular tachycardia should not be influenced by the presence of syncope during the clinical presentation of the arrhythmia.

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