

External and Internal Electrical Cardioversion: Comparative, Prospective Evaluation of Cell Damage by Means of Troponin I

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Introduction and objectives. In this study we measured the concentrations of cardiac troponin I (cTnI) and several biochemical markers of myocardial damage after elective external cardioversion or internal cardioversion by specific catheters or automatic defibrillators.

Material and methods. Biochemical markers were analyzed prospectively for 30 consecutive patients after electrical cardioversion. Concentrations of cTnI, myoglobin, creatine kinase (CK), CK-MB and the MB/CK ratio were determined in samples before cardioversion and 2, 8 and 24 h later. The shock energy ranged from 50 to 360 joules (235±106 joules) in external cardioversions and from 3 to 37 joules (15±8 joules) in internal cardioversions.

Results. We detected abnormal concentrations of CK, myoglobin, CK-MB and MB/CK in 33% of the patients after external cardioversion. The concentrations of cTnI remained within normal limits at all times, with no elevations detected. Whereas no abnormal concentration of any biochemical marker was detected in any patient who required internal cardioversion for atrial fibrillation, two patients who underwent external cardioversion from an automatic defibrillator did have abnormal concentrations of CK-MB, myoglobin, and even of cTnI.

Conclusions. The concentration of cTnI remained below the detection limit after external cardioversion, even though the other more non-specific markers changed. No enzyme alteration was detected in patients who underwent internal cardioversion of atrial fibrillation.

Cardioversión eléctrica externa y sistemas de cardioversión interna: evaluación prospectiva y comparativa del daño celular con troponina I

Introducción y objetivos. En este estudio evaluamos las concentraciones de troponina I cardíaca (cTnI) y distintos marcadores de lesión miocárdica tras cardioversión externa electiva o interna a través de catéteres específicos o de desfibrilador automático.

Material y métodos. Analizamos prospectiva y consecutivamente los datos de 30 pacientes tras cardioversión eléctrica. Se determinaron concentraciones de cTnI, mioglobina, creatincinasa (CK), CK-MB-masa e índice MB/CK en muestras precardioversión, y a las 2, 8 y 24 h poscardioversión. La energía de choque estuvo comprendida entre 50 y 360 J (235 ± 106 J) en cardioversiones externas, y entre 3 y 37 J (15 ± 8 J) en cardioversiones internas.

Resultados. Tras las cardioversiones externas se detectaron valores anormales de CK en el 33% de los pacientes, así como de mioglobina, CK-MB e índice. Las concentraciones de cTnI permanecieron en todo momento dentro de límites normales, sin detectarse elevaciones patológicas en ninguno de los pacientes. A diferencia de las cardioversiones externas, tras las internas, en los pacientes que la precisaron por fibrilación auricular, no se detectaron valores patológicos de ningún marcador bioquímico. Por el contrario, sí encontramos concentraciones patológicas de CK-MB-masa y mioglobina, e incluso de cTnI en dos de los pacientes que recibieron cardioversión interna a través de un desfibrilador automático.

Conclusiones. La concentración de cTnI se mantuvo en valores indetectables tras la cardioversión externa, aunque el resto de los marcadores más inespecíficos se modificaran. En el grupo de cardioversión interna por fibrilación auricular no se detectó ningún movimiento enzimático.

Key words: *Creatine kinase. Atrial fibrillation. Cardioversion.*

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INTRODUCTION

Electrical cardioversion is a critical treatment for the survival of patients with cardiac arrest due to ventricular tachycardia or fibrillation.¹ A large percentage of patients who reach a hospital after

ABBREVIATIONS

cTnI: cardiac troponin I
 CK: creatine kinase activity
 CK-MB: Mb fraction of creatine kinase
 ECv: external electrical cardioversion
 ICv: internal electrical cardioversion
 Cvs: cardioversions

resuscitation from cardiac arrest present elevation of creatine kinase (CK) and its MB fraction (CK-MB).² It is of vital importance for the clinical treatment to define the cause of these elevations of markers of myocardial lesion. Consequently, it is necessary to determine if they are due to myocardial infarction or to damage produced by electrical cardioversion and other resuscitation maneuvers.³ The usefulness of cardiac troponin I (cTnI) in this type of patients lies in its cardiac specificity and, therefore, the fact that it does not rise in skeletal muscle lesions.

In this study we proposed to determine *a*) if electrical cardioversion procedures produce myocardial damage and to quantify it in two clinical electrical cardioversion situations: external, or conventional, cardioversion and internal (either through a specific defibrillation system or through the electrodes of an automatic defibrillator); *b*) the extension of the possible myocardial damage and its correlation with cardioversion variables (energy applied), and *c*) the concentrations and kinetics of cTnI and the different markers of myocardial injury after electrical cardioversion.

MATERIAL AND METHODS

This study included patients referred to the Arrhythmia Unit of our hospital for electrical cardioversion. All patients gave their written consent. All patients who in the preceding month presented myocardial infarction, unstable angina, muscular trauma (including treatment with intramuscular injections), or infectious disease were excluded.

We prospectively and consecutively analyzed the data of 30 patients who underwent electrical cardioversion. The group of external cardioversion was formed by a total of 15 patients (11 men and 4 women). Thirteen of them (11 men and 2 women) underwent scheduled cardioversion for supraventricular arrhythmias (atrial fibrillation in 8 and atrial flutter in 5) and 3 patients (1 man and 2 women) underwent electrophysiological study with programmed pacing who required defibrillation by external electrical cardioversion (ECv) (all 3 for atrial fibrillation). On the other hand, in the group of internal cardioversion a total of 15 patients (10 men and 5 women) were analyzed. In 7 of them (3 men and 3

women) conversion of atrial fibrillation was carried out by means of internal electrical cardioversion (ICv). In these patients ICv was performed because they had been previously refractory to ECv. Finally, in the remaining 8 patients (6 men and 2 women) cardioversion was performed for ventricular defibrillation that occurred during implantation of an automatic defibrillator.

Cardioversion procedures

External cardioversion

ECv was carried out using two different systems: *a*) in 5 patients with two rectangular self-adhesive plates of 8.5 14.5 cm in diameter (one on the right anterior region of the chest and the other on the posterior region) connected to a LifePak defibrillator,⁶ and *b*) in the other 11 patients, with the paddles of the Cardiopak 200 defibrillator, size 7.5 11.0 cm (applied to the right parasternal region and apical region, left lateral mid-axillary area), both covered with conductive gel. The procedure was carried out under mild sedation with propofol (60-200 mg). Discharges of monophasic, exponential and truncated direct current were administered, synchronized with the QRS wave, between two plates or paddles. The initial energy ranged from 100 to 200 J and discharges were repeated until sinus node rhythm was achieved or two maximum energy discharges were delivered (360 J). All shocks were monophasic.

Internal cardioversion

In patients undergoing ICv by a right femoral and left subclavian approach, after percutaneous puncture under local anesthesia with 1% lidocaine and insertion of 7-Fr introducers, two different systems were inserted: one with two broad helicoid electrodes of 5.5 cm and 5 cm² area (Vascostin Tc, InControl), to the lateral region of the right atrium and coronary sinus, and another single-catheter system with two electrodes for defibrillation, one positioned in the left pulmonary artery and the other in the right atrium (Catheter Alert, EP-MedSystems). The electrodes were connected to an external defibrillator capable of delivering biphasic, low-energy discharges. The shocks were administered with progressively greater energies from 2 to 15 J. The procedure was carried out under mild sedation with propofol (60-200 mg). The shocks were always monophasic.

The defibrillator was implanted in the electrophysiology laboratory. Percutaneous puncture of the subclavian vein was performed, leaving a 0.2-mm J guide wire introduced in the right atrium under fluoroscopic control. An incision was made in the left

pectoral region, below the clavicle. Later a pocket was prepared under the fascia of the greater pectoral muscle. The sensing and defibrillation electrode was introduced into the apex of the right ventricle through a 10.5-Fr introducer. The electrodes were mostly passive fixation electrodes (Medtronic or CPI), except for those used with the Medtronic Transvene system (active fixation). Anesthesia with intravenous propofol was administered (60-200 mg dose, based on weight and age). A first low-energy (0.5-1.5 J) internal discharge was applied synchronized with the QRS wave to verify the integrity of the circuit and its impedance. Then ventricular fibrillation was induced, either by a shock on T or with alternating current. In the first shock energies between 15 J and 20 J were used and reduced progressively until reaching the threshold or achieving an acceptable minimum defibrillation energy (safety margin of more than 10 J). These shocks were always biphasic.

In all patients, after each procedure any change in the clinical state, modifications of the ST segment, and the appearance of new arrhythmias were recorded.

Markers of myocardial injury

We determined and compared serum concentrations of CK-MB mass, cTnI, myoglobin, and CK activity. We also calculated the CK-MB mass index as a percentage (index) of total CK activity. For serum determinations of markers, 5-ml samples of peripheral blood were collected in tubes without anticoagulant in accordance with the following scheme: pre-cardioversion extraction (baseline) and extractions 2 h, 8 h, and 24 h after the procedure. All samples were centrifuged within 60 min of collection. Part of the markers were processed in fresh samples, and the rest of the sample was divided in aliquots that were frozen at -20°C until processing. The mean time before the determination in frozen samples was 28 ± 7 days, which was carried out after thawing aliquots to room temperature, then performing the measurements within 1 h.

From a fresh aliquot of the baseline sample, a biochemical study was made that included the determination of glucose, creatinine, urea, sodium, potassium, and aspartate aminotransferase activity. Routine Hitachi 747 analyzer techniques were used (Roche Diagnostics). In all fresh samples CK activity and the concentrations of myoglobin and CK-MB mass were determined. CK activity was determined with an Integra 700 analyzer (Roche Diagnostics). The rest of the markers (CK-MB mass and myoglobin) were determined using automatic immunoanalysis techniques with double monoclonal antibody and detection by chemoluminescence in an ACCESS analyzer (Izasa-Beckman). The cTnI was determined in the frozen aliquots using immunological

«sandwich» techniques with double monoclonal antibody in an automatic enzymofluoroimmunoanalysis (with radial bipartition) in a Stratus II analyzer (Dade-Behring). The cutoff points of our laboratory for the markers studied were: CK >200 U/L, CK-MB mass >5 $\mu\text{g/L}$, index $>5\%$, myoglobin >80 $\mu\text{g/L}$, and cTnI >0.8 $\mu\text{g/L}$.

Statistical analysis

The continuous variables were expressed as the mean and 95% confidence interval (CI) or associated limits when the distributions were normal, after analyzing the distribution of frequencies by means of the Kolmogorov-Smirnov test. The Wilcoxon test was used in variables without a normal distribution, and their results were expressed as the median and interquartile intervals. To determine statistical differences between continuous variables, the Student t test was used. A value of $P<.05$ was considered significant. The correlations between variables were made by linear regression (Pearson). This statistical analysis was carried out with the SPSS 10.0 statistical application and Excel 2000 spreadsheet.

RESULTS

This study finally included a total of 21 men and 9 women with a mean age of 58 ± 14 years and range of 25 to 81 years (Table 1), without statistically significant differences between sexes (mean \pm standard deviation: 58 ± 15 years and 59 ± 13 years, for men and women, respectively). Patient weight ranged from 62 to 95 kg (77.3 ± 10.6 kg); height 152 to 177 cm (164.8 ± 7.4 cm), and body mass index 25.3 to 32.9 (28.1 ± 1.9), with statistically significant differences between men and women ($P<.05$).

The 30 patients analyzed underwent 73 electrical discharges with direct current. No complications were observed during or after cardioversion in any patient, nor were changes in the ST segment detected. In all patients, conversion of the arrhythmia was achieved and 24 of the 25 patients remained in sinus rhythm after 24 h. With respect to the number of cardioversions, cumulative energy and maximum energy applied, we found significant differences between external cardioversion (with a smaller number and higher energy) and internal cardioversion (larger number and lower energy). The energy of each shock ranged from 50 to 360 J (235 ± 106 J) in external cardioversion, and from 3 to 37 J (15 ± 8 J) in internal cardioversion. It should be emphasized that in the internal cardioversion group, although there were no significant differences in the maximum energy applied, we observed a larger number of cardioversions and greater cumulative energy in patients with automatic defibrillator (4 cardioversions

TABLE 1. General characteristics of patients undergoing electrical cardioversion

	Total	ECv	ICv
No., m/f	30 (21/9)	15 (11/4)	15 (10/5)
Age, y	58±14 (25-81)*	56±17 (25-81)	61±8 (47-73)**
Weight, kg	77±11 (62-95)*	78±17 (65-95)	74±8 (62-89)**
Height, cm	165±7 (152-177)**	166±8 (156-177)	165±9 (152-175)**
Body mass index, kg/m ²	28.5±1.9 (25.3-32.9)*	28.3±2.1 (26.0-32.9)	27.2±2.0 (25.3-31.3)**
Organic heart disease, No. (%)	22	10 (66)	12 (80)
Ischemic heart disease, No. (%)	12	5 (33)	7 (47)
Diabetes mellitus, No. (%)	4	3 (20)	1 (7)
Bundle-branch block, No. (%)	7	3 (20)	4 (27)

Results are presented as mean±standard deviation (limits). N indicates number of patients; Ecv, group of external electrical cardioversion; Icv, group of internal electrical cardioversion. *P<.05 between men and women; **Nonsignificant compared with the ECv group.

and 76 J versus 3 cardioversions and 25 J for internal defibrillators and internal cardioversion, respectively). In this respect, there were no differences between types of external cardioversion (plates and paddles).

Markers of myocardial lesion after external cardioversion

The different parameters evaluated in the routine biochemical study of the baseline sample presented values within normal reference limits in 14 patients. Only some of these parameters were altered in a female patient diagnosed as kidney failure (CK, 3.41 mg/dL; urea, 142 mg/dL, and urates, 8.2 mg/dL).

Initially, all the patients had normal values of CK activity and CK-MB mass and cTnI concentrations. Only the patient with kidney failure had high baseline myoglobin concentrations and CK-MB/CK index (>5%), these markers being normal in the rest of the patients. In 60% of the patients who underwent external cardioversion, none of the markers increased (Table 2). After external cardioversion, increased CK activity was detected in 33% of the cases (5 patients). In the same patients abnormal myoglobin

concentrations were found, and in two of them, abnormal CK-MB mass concentration. These 5 patients had received at least one defibrillation shock at an energy over 300 J; in addition, they required cumulative energies of more than 500 J, which was statistically significant (P<.05) with respect to the rest of the patients in this group (580±50 J vs 205±106 J, respectively). One of these patients presented the highest concentration of CK-MB mass (23.3 ng/mL) and extremely pathological CK (21 428 U/L) and myoglobin values (3948 ng/mL). In addition to these 5 patients, myoglobin rose to abnormal concentrations in the patient with chronic kidney failure. Although the baseline values were abnormal (170 ng/mL), CK reached a maximum value of 293 ng/mL in 8 h. In this same patient, a pathological peak CK-MB mass concentration was detected but the elevation of CK activity did not reach the cutoff point. No differences were found in the release of these markers between the two types of external cardioversion used in this study (plates and paddles).

In contrast with the rest of the markers, cTnI concentrations remained within normal limits, and no pathological elevations were detected in any patients.

TABLE 2. Markers of myocardial lesion after external and internal electrical cardioversion

		External		Internal	
		Baseline	Peak	Baseline	Peak
CK		76±40 (26-165)	618±1.200 (34-21.428)	66±33 (33-139)	181±149 (43-453)
(cutoff point: 200 UI/l)	N (%)	0 (0)	5 (33)	0 (0)	4 (27)
CK-MB mass		1.6±1.1 (0.1-4.0)	2.6±1.9 (0.5-23.3)	1.5±0.9 (0.5-3.3)	2.8±1.8 (0.9-6.5)
(mean±SD [limits])					
(cutoff point: 5.0 g/l)	N (%)	0 (0)	3 (20)	0 (0)	2 (13)
Index (mean±SD [limits])		2.4±2.1 (0.4-9.3)	3.0±1.1 (1.1-9.3)	2.2±1.3 (0.3-4.6)	3.0±2.1 (0.5-8.3)
(cutoff point, 5%)	N (%)	1 (6)	1 (6)	0 (0)	1 (6)
Myoglobin (mean±SD [limits])		42 ± 47 (5-170)	199 ± 330 (9-3.948)	32 ± 14 (20-58)	80 ± 51 (23-167)
(cutoff point: 80 g/l)	N (%)	1 (6)	6 (40)	0 (0)	4 (27)
cTnI (mean±SD [limits])		0.0±0.1 (0.0-0.3)	0.2±0.2 (0.0-0.4)	0.0±0.0 (0.0-0.1)	0.4±0.5 (0.0-1.3)
(cutoff point: 80 µg/l)	N (%)	0 (0)	0 (0)	0 (0)	2 (13)

SD indicates standard deviation; N, number of patients with values above the cutoff point (percentage of total); CK, creatine kinase; cTnI, cardiac troponin I. *P<.05 compared with external cardioversion.

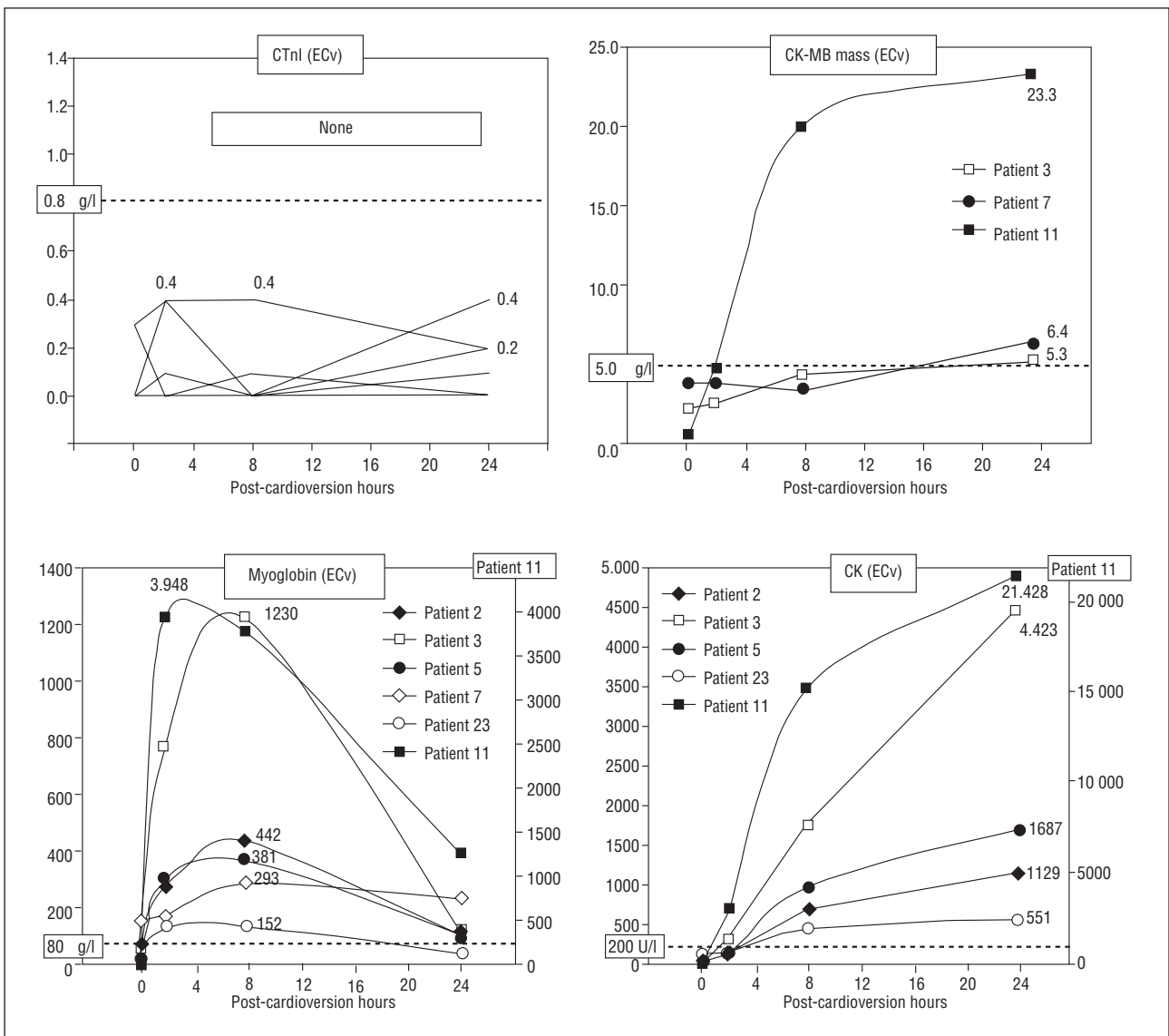


Fig. 1. In this figure, the markers of myocardial lesion (markers) in patients in the external electrical cardioversion group (ECv) that surpassed the cutoff point of the marker in any extraction are shown. The concentration of the marker in each extraction, peak value reached, and cutoff point are shown in relation to time (hours) after the procedure. In the case of cardiac troponin I (cTnI), no patient had values above the limit of detection of the technique (0.4 µg/l). Two patients presented slight elevations in CK-MB mass at 24 h, whereas a third patient had a clear elevation from 2 h on, although this patient also reached the peak value at 24 h. This same patient (patient 11), who had extremely pathological myoglobin and creatinine (CK) values, is shown on a secondary Y axis in the graphs of these markers for purposes of illustration. With regard to myoglobin, it should be noted that patient 7 (diagnosed as kidney failure) was the only patient with pathological baseline values; nonetheless, he experienced a clear increase in this marker (from 170 to 293 µg/l).

Not only were no abnormal values detected, but in the 15 patients in our study cTnI concentration constantly remained below the limit of detection of the technique (0.4 ng/mL) in all post-cardioversion extractions (Table 2). As far as the kinetics of the markers (Figure 1), it must be emphasized that the peak values of the earliest markers were reached in 8 h (myoglobin) and 24 h (CK-MB mass) in most patients (13 of 15).

Correlations in external cardioversion

As far as the results of the analysis of the linear correlation between the peak concentrations of markers and the cardioversion variables, excellent coefficients of correlation were obtained ($r > 0.9$) between the peak values of CK, CK-MB mass, and myoglobin for each patient and the number of cardioversions. The coefficients of correlation between the peak values of the same markers and the total

energy applied were also higher than 0.9. No correlation was found between the values of cTnI and the CK-MB/CK index and the number of cardioversions and total energy applied; likewise, no correlation was found between any of the markers studied and the maximum energy reached. CK and myoglobin presented the best correlation ($r=0.970$) of the correlations between peak values of different markers.

Markers of myocardial lesion after internal cardioversion

As in the ECv group, in 73% of the patients (11 of 15) who underwent ICv, none of the markers studied rose to pathological values. After ICv, maximum pathological concentrations were detected in 4, 2, 1 and 4 of the patients of this group for CK activity, CK-MB mass concentration, CK-MB mass to CK activity index, and myoglobin concentration, respectively. These percentages of patients with pathological results were similar to those obtained after ECv for the same markers (Table 2). Nevertheless, unlike ECv, after ICv we observed cTnI concentrations above the cutoff point in two patients, associated with pathological elevation of CK-MB mass and myoglobin. These two patients were the two men who underwent defibrillator implantation and received the largest number of electrical discharges: 14 and 7, versus a median number (range) of 4 (3 to 6) cardioversions in the other 10 patients. In one of them (the one who received the largest number of cardioversions, 14, as well as the greatest cumulative energy in the ICv group, 188 J), CK activity was also elevated, but not the CK-MB/CK index. In contrast, the CK-MB/CK index rose in the other patient, but CK activity did not surpass the cutoff point in any extraction. We found pathological elevations of CK activity in 3 more patients in this group, all of whom had an implanted defibrillator. In 2 of these 3 patients the myoglobin concentration also rose above the cutoff point, whereas in the third the concentration rose but remained within the limits of normality (baseline myoglobin: 35 ng/mL, peak myoglobin: 75 ng/mL). The myoglobin concentrations and observed CK activity after ICv were significantly lower ($P<.05$) than in the ECv group. Except in the 2 patients mentioned previously, cTnI concentrations always remained below the limit of detection of the technique in all the extractions of the other patients except one. In this patient (a man with an implanted defibrillator, 6 cardioversions, and the second highest cumulative energy received, 152 J), a cTnI concentration of 0.7 ng/mL was reached at 8 h, but the variations in the concentrations of the rest of the markers remained within the limits of normality.

The 7 patients in the ICv group who underwent ICv

for atrial fibrillation were the patients with the lowest number of cardioversions (1.5 ± 0.9) and lowest cumulative energy (17.8 ± 6.9 J versus a mean of 76 J in the 8 patients with defibrillator implants). It is notable that in these 7 patients none of the markers studied reached abnormal values.

With regard to kinetics, in the ICv group the peak values of the earliest markers (myoglobin and CK-MB mass) were reached in an earlier extraction (2 and 8 h post-cardioversion, respectively) than in the ECv patients in most cases. The peak cTnI value occurred 8 h post-cardioversion in the only 3 patients in which cTnI concentration changed. It should be emphasized that the 2 patients in which myoglobin rose without an elevation in cTnI and CK-MB mass presented peak values at 8 h instead of 2 h as in the patients in which cardiospecific markers did rise.

Correlations in internal cardioversion

The best correlations between peak marker values, the number of cardioversions, and cumulative energy were obtained with the most cardiospecific markers (cTnI and CK-MB mass; 0.798 and 0.734, respectively). We found no correlation between any of the markers and the maximum energy reached (all less than 0.2). The correlation of maximum concentrations of the different markers indicated that, as for ECv, the best correlation obtained was between CK activity and myoglobin ($r=0.976$) and that CK-MB mass seems to correlate better with these markers in ICv.

DISCUSSION

Since Beck established in 1947 the basis for the emergency treatment of cardiac arrest and ventricular fibrillation by external application of continuous electrical current, this has been introduced in clinical practice for the conversion of tachyarrhythmias.⁴⁻⁶ Although the method has been used routinely for 40 years, there is still controversy as to whether applications of direct current can cause myocardial damage.^{7,8} Since the 1970s it has been known that multiple and repeated electrical cardioversions can affect myocardial function, 9 produce transitory ST-segment elevations, 10 and induce the release of enzymes and muscle proteins habitually used as markers.¹¹

The application of discharges of direct current can cause reversible damage of subcellular structures involved in oxidative phosphorylation¹² and originate a variable release nonspecific proteins of striate cardiac muscle, including CK, CK-MB, and myoglobin, which makes the diagnosis of a previous acute myocardial lesion difficult.^{11,13,14} These biochemical markers are not cardiospecific, which is why the skeletal muscle origin of the post-

cardioversion elevations is accepted, as a result of the faradic effect of the current applied.¹² However, experimental studies in animals have offered histological evidence of localized myocardial necrosis.^{15,16} However, CK activity and CK-MB and myoglobin concentrations fail to reveal minimal myocardial lesions, especially if simultaneous skeletal muscle damage also exists.¹⁷ The recent availability of cardiospecific markers like cTnI, which are not expressed in skeletal muscle, has made possible a more a more effective discrimination of the skeletal or myocardial origin of muscle lesions,¹ as well as the detection of minimal myocardial damage that takes place in certain clinical and experimental situations.¹⁸⁻²⁰

External electrical cardioversion

In our study, cTnI concentrations always remained undetectable after cardioversion. In contrast, we observed pathological elevations in CK activity, CK-MB mass, and myoglobin, respectively, as in previous studies.^{12,13} The origin of these elevations, given that they are not accompanied by pathological cTnI concentrations, seems to be muscular. Since the studies of Resnekov and McDonald in the late 1960s,²¹ it is known that the peripheral skeletal muscular damage is the most common adverse effect of ECv. Injury of the chest wall skeletal muscle, which produces release of noncardiospecific markers after cardioversion, has been clearly demonstrated by histopathological studies²² and imaging techniques with radionuclide uptake.²³ These disturbances can lead to disruption of the cell membrane, with rupture and release of intracellular components like cytosolic proteins.²⁴

In our series of patients, an excellent correlation was demonstrated between the elevation of noncardiospecific markers (CK activity and myoglobin), the number of cardioversions, and the total amount of applied energy, with coefficients of correlation of more than 0.9. As for the correlation between markers after ECv, there was a magnificent correlation between peak CK and myoglobin values, scant correlation between these markers and CK-MB mass ($r < 0.5$ for both), and no correlation with cTnI.

Some studies have reported slight elevations in troponins, cTnI in a small percentage of patients (3 of 38) and with low values (0.8-1.5 ng/mL), 13 as well as cTnT, 25 after repeated ECv for ventricular arrhythmias in patients undergoing electrophysiological study. We did not observe such elevations in our study, probably because the converted arrhythmias were supraventricular and no patients with ventricular tachycardia were included.

Internal electrical cardioversion

In the ICv group, we observed elevation of the cTnI concentration and other markers to pathological values in two patients who underwent defibrillator implantation. In addition, in a third patient cTnI elevation to detectable values (0.7 ng/mL) occurred, but did not reach the cutoff point. These elevations took place in the patients who received more total energy and greater number of cardioversions. In the rest of the patients, the cTnI remained undetectable and CK-MB mass did not reach pathological values.

After repeated ICv during defibrillator implantation, we observed abnormal CK and myoglobin concentrations without elevation of the most cardiospecific markers in other patients. These elevations must be induced by the same circumstance as those detected in electrical cardioversion. In the case of ICv, these elevations are smaller, because less energy is used and this energy is transmitted to skeletal muscle in a more indirect way.

In the 4 patients who underwent ICv in our study, we did not detect any variation in marker values. In these patients we used fewer cardioversions and applied less total energy, since it is a very effective technique for achieving conversion to sinus node rhythm of patients with atrial fibrillation in which the ECv technique has failed for different reasons.²⁶

It should be remembered that the cTnI titers reached (around 1.0 ng/mL) are far from those documented after extensive myocardial lesions, like those of myocardial infarction, or even small lesions, like those produced by intracardiac radiofrequency catheter ablation.^{19,20} In contrast with these lesions, cTnI concentrations after ICv present normal values 24 h post-cardioversion. This type of damage is very similar, as much in the concentrations reached as in their kinetics, to that observed in electrophysiological studies by catheterization.¹⁸ The endomyocardial catheters used in defibrillator implantation are thicker (as much as 10.5 Fr or more), although more flexible and ductile than those usually in ablation, electrophysiological studies, or ICv; in addition, they need to be affixed to the ventricular myocardium.

On the other hand, the CK and myoglobin values reached after ICv in our study are clearly lower than those reached after ECv. Consequently, it seems that ICv is capable of converting arrhythmias, with minimal myocardial damage in a low percentage of patients, and produces less skeletal muscle damage than ECv.

Usefulness of cardiac troponin I in emergent cardioversion

The combined effects of prolonged cardiopulmonary resuscitation and multiple cardioversions have not yet been properly established, which is why caution is necessary when extrapolating these results to this type of patients. As can be seen in

our study, ECv does not produce any type of myocardial lesion and the pathological elevations of CK and CK-MB often observed in patients who undergo this procedure are of skeletal muscle origin. ICv can produce minimal myocardial damage when applied with endomyocardial catheters to the ventricular face. This is one of the contributions of this article, a comparison of the markers of myocardial lesion in ECv and ICv, which helps to understand the meaning of elevations in markers in these situations.

The methodological limitations of the study include the absence of randomization in the selection of patients in each group and, therefore, the possibility of selection bias in the assignment of cases to the two cardioversion techniques compared.

CONCLUSIONS

After ECv, no myocardial lesion occurs that can be detected by means of cTnI analysis, but skeletal muscle lesions occur that correlate with the number of continuous current discharges applied and the total energy delivered. Internal cardioversion through automatic defibrillator electrodes can originate minimal myocardial damage that becomes undetectable after 24 h. In the ICv group with the specific system used for conversion of atrial fibrillation, no variation in enzyme levels was detected.

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