Serum enzyme activities after cardioversion

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Serum aspartate aminotransferase (SGOT), alanine aminotransferase (SGPT), creatinine phosphokinase (CPK), and butyric acid dehydrogenase (BDH) were determined in 94 patients before, 1½ hours, and 24 hours after cardioversion. An increase in SGOT and CPK activity was observed 24 hours after cardioversion in the group of patients treated with two or more DC shocks. The importance of this enzyme activity increase is discussed. It originates in the skeletal muscles and probably has no clinical significance, as no other signs of myocardial damage were observed simultaneously in a large group of patients.

Cardioversion has an established place among the methods of treatment of cardiac arrhythmias. The method is generally safe. However, some complications due to this technique may occur, including systemic emboli, cardiac arrhythmias, and ST and T changes in the electrocardiogram (Åberg and Cullhed, 1968; Lown, 1967; Resnekov and McDonald, 1967, 1968; Sussman, Woldenberg, and Cohen, 1964). Some authors have noted an increase of serum indicator enzyme activities after cardioversion. This was interpreted as caused either by myocardial or by skeletal muscle damage, or as a phenomenon without any clinical significance. The reports published hitherto have been based on small groups of patients. The aim of the present contribution was to study this phenomenon in a larger group of patients.

Methods and subjects

The cardioversion was performed using the DC defibrillator (Godart), always under light general barbiturate or Epontil anaesthesia. The energy discharged during the first electroshock was 150 watt sec., and, if necessary, was gradually increased during every subsequent electroshock to 400 watt sec. One electrode was placed under the right clavicle and the other in the left middle axillary line in the fifth intercostal space.

One blood sample for the determination of aspartate (SGOT) and alanine (SGPT) aminotransferases, creatine kinase (CPK), and butyric acid dehydrogenase (BDH) was drawn before cardioversion, the second 1½ hours, and the third 24 hours after direct-current countershock. Serum activity of aminotransferases was determined according to Reitman and Frankel (1957). The upper normal limit for this method in our labora-

tory is 30 I.U. The serum activity of CPK was examined by the method of Hughes (1962) (the upper normal limit for men 3.9 U., for women 2.7 U.), and serum BDH activity by the method of Rosalki (1962) (upper normal limit 131 U.).

The statistical analysis of the data was made by the paired experiment method (a modification of Student's test) (Oktaba, 1966).

A total of 94 patients treated with DC shock were studied. SGOT and SGPT activities were determined in 81 of them, CPK in 59, and BDH in 54. The age of the patients varied between 22 and 73 years. Valvular heart disease was diagnosed in 51 patients, atherosclerotic heart disease in 38, lone fibrillation in 4, and treated thyrotoxicosis in 1 patient. The reason for cardioversion was atrial fibrillation in 70 cases, atrial flutter in 19, supraventricular paroxysmal tachycardia in 4, and ventricular paroxysmal tachycardia in 1 case. Cardioversion restored the normal sinus rhythm in 83 cases, while in 11 cases it was ineffective.

In order to evaluate the relation between the amount of electrical energy used and the enzyme activity changes, the patients were divided into two groups. Group I, treated with one countershock (Table 1), and Group II, those treated with two or more countershocks (Table 2).

Results

The results are shown in Tables 1 and 2. In the first group (Table 1) the activity of all four enzymes changed only slightly after cardioversion. In the second group (Table 2) a statistically significant increase in the SGOT ($t=4.85$, $p<0.01$) and CPK ($t=2.51$, $p<0.05$) activities was observed 24 hours after cardioversion. In spite of this statistical significance, the increase in SGOT activity was not great and often occurred within the normal range. It was higher than the upper limit only in 6 out of 25 patients. The highest SGOT activity in this group of patients was 48 I.U.

On the other hand, the increase of CPK activity (the highest value 63 U.), though less significant, was higher than the upper limit in 8 out of 16 cases (Fig. 1).

We have never seen ST and T changes in the electrocardiograms of the patients with increased enzyme activity, though we have observed such changes in other patients treated with cardioversion in our Clinic (Mandecki *et al.*, 1969). Neither have we observed any complications in this group, except the transient cardiac rhythm disorders usually encountered after cardioversion, which hardly ever need pharmacological treatment.

Discussion

Many authors have reported an increase in the muscle enzyme activity after cardioversion (Åberg and Cullhed, 1968; Caesar *et al.*, 1969; Castle and Hecht, 1964; Hunt and Bailie, 1968; Konttinen *et al.*, 1969; Reinikainen *et al.*, 1965; Slodki *et al.*, 1966; Sussman *et al.*, 1964; Turner and Towers, 1965), most often an increase in SGOT and CPK activity. This is also supported by our study. In our subjects the activity of these two enzymes rose only in the group of patients who were treated with two or more DC countershocks. This indicated that a relation existed between these changes and the amount of the electrical energy applied. The other reports on this problem agree (Caesar *et al.*, 1969; Castle and Hecht, 1964; Turner and Towers, 1965), though some authors did not find this relation (Åberg and Cullhed, 1968; Slodki *et al.*, 1966).

The lack of complications as well as of electrocardiographic abnormalities in the patients with increased enzyme activity may suggest that the phenomenon we have observed is of no clinical significance. Lown *et al.* (1962) and Peléska and Zastava (1966) showed the possibility of morphological and electrocardiographic changes in animals after countershock, especially when alternating current was used. But most authors investigating this problem think that the changes in enzyme activity are not caused by myocardial injury. A case treated with 140 countershocks was reported (Kong and Proudfit, 1964), without any myocardial changes caused by electric current at necropsy. The increase in enzyme activity may be due to the enhanced cellular membrane permeability following the action of electric current, and this may facilitate the release of the cellular enzymes into the blood-stream (Slodki *et al.*, 1966).

The normal SGPT activity in our patients

TABLE 1 Patients treated with 1 DC shock

Enzyme	No. of patients	Enzyme activity (mean values)		
		Before cardioversion	1½ hours after cardioversion	24 hours after cardioversion
SGOT	57	17.9	18.9 $t=0.56$ $p>0.05$	19.3 $t=0.75$ $p>0.05$
SGPT	57	26.9	26.4 $t=0.26$ $p>0.05$	29.2 $t=0.71$ $p>0.05$
CPK	43	3.01	2.74 $t=1.27$ $p>0.05$	2.96 $t=0.14$ $p>0.05$
BDH	34	117.9	119.1 $t=0.12$ $p>0.05$	116.9 $t=0.13$ $p>0.05$

TABLE 2 Patients treated with 2 or more DC shocks

Enzyme	No. of patients	Enzyme activity (mean values)		
		Before cardioversion	1½ hours after cardioversion	24 hours after cardioversion
SGOT	25	13.8	13.4 $t=0.31$ $p>0.05$	19.8 $t=4.85$ $p>0.01$
SGPT	25	16.8	14.7 $t=1.29$ $p>0.05$	18.3 $t=0.60$ $p>0.05$
CPK	16	2.17	2.20 $t=0.32$ $p>0.05$	11.65 $t=2.51$ $p<0.05$
BDH	20	97.0	101.8 $t=0.58$ $p>0.05$	88.2 $t=0.97$ $p>0.05$

FIG. Serum CPK after cardioversion (the patients with two or more DC shocks).

seems to exclude the liver as the source of other altered enzyme activities. The most probable source of the increased SGOT and CPK activity is skeletal muscles, where these two enzymes are abundantly present. Other authors are of the same opinion (Caesar *et al.*, 1969; Hunt and Bailie, 1968; Konttinen *et al.*, 1969). Recently, Konttinen *et al.* (1969), in their study on LDH isoenzyme activity after cardioversion, excluded the myocardium as the enzyme-releasing tissue. Slodki *et al.* (1966) reported that aldolase, known to indicate skeletal muscle damage (Hughes, 1962), increased more sensitively than SGOT after DC shock. Raised serum CPK activity does not necessarily indicate necrosis, since muscular exercise (Griffiths, 1965) and also appendectomy (Hunt and Bailie, 1968) can increase serum CPK activity. An increased tissue permeability evoked by hyperaemia (the thermoelectric effect) or contractions of thoracic muscles may cause the release of enzymes into the serum after cardioversion (Konttinen *et al.*, 1969).

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