Determinants of Elevated Creatine Kinase Activity and Creatine Kinase MB-Fraction following Cardiopulmonary Resuscitation*

Joseph Mattana, M.D.; and Pravin C. Singhal, M.D.

Objective: We undertook this study to determine the occurrence and the determinants of elevation of serum creatine kinase (CK) levels and CK MB-fraction following cardiopulmonary resuscitation (CPR).

Design: Four hundred twenty consecutive adult admissions to the Long Island Jewish Medical Center from January 1989 through December 1990 with a diagnosis of cardiac arrest were reviewed.

Setting: The Long Island Jewish Medical Center, New Hyde Park, NY, the Long Island Campus for the Albert Einstein College of Medicine, Bronx, NY.

Patients: Sixty-three patients survived for at least 12 h following cardiac arrest for evaluation of post-CPR CK levels and were included into the study.

Measurements: Clinical features, biochemical profiles, and administered drug profiles were studied in these patients. The clinical and biochemical features of the patients with (CK >224 IU/L [3.7 µkat/L]) and without rhabdomyolysis were also compared.

Main Results: Two major determinants responsible for elevated CK levels emerged, including physical injury (number of chest compressions during CPR) and electrical injury (cumulative number of joules administered during defibrillation). Post-CPR CK levels showed positive correlations with both the number of chest compressions given (p<0.001) and the number of joules administered during defibrillation (p<0.001). Post-CPR CK-MB levels also showed a positive correlation with the number of joules administered (p<0.005) and the number of chest compressions (p<0.02). Forty-three (68.3 percent) of the 63 patients developed rhabdomyolysis. Serum CK levels were higher (p<0.005) in the patients who received electrical countershock therapy as well as chest compressions when compared with patients who received chest compressions alone. There were no significant differences in electrolyte levels between patients with and without rhabdomyolysis. Thirty patients had a history of coronary artery disease (CAD) and 18 (60.0 percent) of these had a positive MB-fraction post-CPR while only ten of the 33 patients without known CAD had a positive MB-fraction post-CPR (30.3 percent, p<0.05). Patients with no known CAD but positive CK-MB fraction had significantly higher total CK levels, physical injury, and electrical injury compared with patients with negative CK-MB fraction.

Twenty patients survived CPR and were discharged from the hospital without significant neurologic sequelae. The remaining 43 either died or suffered severe neurologic injury. The patients who survived CPR had a significantly shorter duration of CPR (p<0.01) compared with those who did not. Patients who did not have long-term survival following CPR were more likely to have elevated serum potassium, phosphate, and creatinine values.

Conclusions: CK elevation is a common finding following successful CPR after cardiac arrest and this elevation of post-CPR CK levels is related to both physical as well as electrical injury sustained during CPR. Elevation of post-CPR CK-MB fraction seems to be only a crude indicator of preexisting CAD; however, a positive CK-MB fraction in patients without CAD is related to severity of physical injury and electrical injury during CPR. Patients who survive CPR without neurologic impairment appear to be those with a shorter duration of CPR. Elevated serum potassium, phosphate, and creatinine values may be related to an adverse effect on long-term survival.

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Increased levels of creatine kinase activity (CK) are commonly observed following cardiopulmonary resuscitation (CPR). There are often multiple causative factors present to account for increased CK levels, including myocardial infarction, physical and electrical injury secondary to chest compressions and defibrillation, impaired tissue perfusion secondary to hypotension and perhaps vasoconstriction from epinephrine, electrolyte disorders such as hypokalemia, hypophosphatemia, and hyperosmolar states and others. In many patients surviving CPR, an elevation of serum creatine kinase MB fraction (CK-MB) is also detected. In some it is believed to be secondary to acute myocardial infarction (eg, as the precipitating event for cardiac arrest), trauma to the myocardium from chest compressions and or defibrillation, or from diminished coronary arterial blood flow from hypotension possibly superimposed on underlying coronary artery stenosis.

An elevation of MB fraction of CK due to cardiac burn following cardioversion has been reported invariably with the use of alternate current countershocks. However, the occurrence of cardiac injury or an

*From the Department of Medicine, Long Island Jewish Medical Center, New Hyde Park, NY, and the Long Island Campus for the Albert Einstein College of Medicine, Bronx, NY.

Manuscript received October 1; revision accepted November 18. Reprint requests: Dr. Singhal, Nephrology Division, Long Island Jewish Medical Center, New Hyde Park, New York 11042.

CAD = coronary artery disease; CK = creatine kinase; CK-MB = MB fraction of CK enzyme; CPR = cardiopulmonary resuscitation; EMS = emergency medical service; LVH = left ventricular hypertrophy.
elevation of MB fraction of CK has been demonstrated to be unusual after the use of direct current counter-shocks. Therefore, it was suggested that an elevation of MB fraction of CK after direct current countershock should be suggestive of cardiac ischemia rather than electric current-related cardiac injury. None of these studies evaluated the combined effect of chest compressions and direct current countershocks on CK enzyme levels.

Recently, Ingwall et al reported that CK-MB fraction is almost nonexistent in biopsy specimens of normal human left ventricular myocardium but is found only in myocardium from patients with coronary artery disease (CAD) and/or left ventricular hypertrophy (LVH). They suggest therefore that patients with mild or no CAD would not be expected to have increased CK-MB present in the serum following myocardial infarction. If it is true that CK-MB is present only in abnormal myocardium, then one would expect a patient with CAD to be much more likely to have a positive CK-MB fraction following CPR. Furthermore, if this is true, then patients without known CAD who have CK-MB present following CPR may warrant an evaluation for CAD.

We undertook the present study to evaluate the relationship between physical and electrical injury during CPR and post-CPR CK and CK-MB activity in patients with and without CAD. We have also made an attempt to determine the major determinants for elevation of CK and CK-MB following CPR. In addition, we have also compared and contrasted clinical and biochemical features between patients surviving CPR and patients dying in the hospital after CPR or who suffered severe neurologic impairment after CPR.

METHODS

Patients

We studied 420 consecutive adult admissions to the Long Island Jewish Medical Center from January 1989 through December 1990 with a diagnosis of cardiac arrest either at the time of admission or at some point during their hospitalization. Of these patients, 63 survived for at least 12 h or more following cardiac arrest and had postcardiac arrest CK levels sent. For each patient we reviewed the events during CPR and determined the duration of chest compressions in minutes and multiplied this by 60 compressions per minute to obtain the approximate number of compressions administered and compared these values with postcardiac arrest CK levels. For each of the 31 patients who underwent defibrillation or cardioversion, we determined the cumulative number of joules administered and compared this with postcardiac arrest CK values and CK-MB values. When chest compressions or defibrillation were performed by emergency medical service (EMS) personnel prior to hospital arrival, we included these data in calculating cumulative chest compressions and joules. We also recorded all medications administered during CPR, including any given by EMS personnel.

To determine a relationship between CK-MB fraction and CPR, we also recorded the presence of CK-MB fraction and compared this in patients with and without CAD. Coronary artery disease was determined to be present if there was a history of angina, myocardial infarction, positive stress testing, and or angiographically proven coronary artery lesions.

The patients were also divided into two groups: patients who showed biochemical evidence of rhabdomyolysis or cardiomyolysis (serum CK levels >224 U/L [3.7 µkat/L]) were included in group 1 and patients without rhabdomyolysis (serum CK <224 U/L [3.7 µkat/L]) were included in group 2. We compared and contrasted clinical and biochemical features in the patients with and without rhabdomyolysis.

Patients were also divided according to long-term outcome of CPR. Group A included patients surviving CPR and who were discharged from the hospital without neurologic impairment and group B included those patients who either died in the hospital or suffered severe neurologic impairment following CPR. Clinical and biochemical features were compared in these groups, including age, duration of CPR, and serum CK, CK-MB fraction, potassium, and phosphate levels post-CPR.

Tissue hypoxia from poor perfusion during cardiac arrest was considered to be a variable in CK elevation. However, all patients were being actively resuscitated during this time of presumed tissue hypoxia and it was difficult to reliably assess the adequacy of perfusion for many of these patients. Thus, we have not made an attempt to correlate tissue hypoxia with CK levels.

Statistical Analyses

Comparison of serum values and other variables between the two groups was carried out by using the unpaired t test. To evaluate the linear association between two variables, scatter plots were drawn and the regression coefficient was calculated. To determine the significance of having or not having CAD in these patients, we applied the χ2 test.

RESULTS

For all 63 patients, ages ranged from 24 to 95 years with a mean age of 65.6 ± 1.93 years. Of these patients, only 22 survived to be discharged from the hospital and two of these patients had suffered severe neurologic damage as a consequence of the arrest. Two other patients survived for 4 and 6.5 months, respectively, but had no evidence of cortical brain function. Postcardiac arrest peak CK activity ranged from 17 to 13,140 U/L (0.28 to 219 µkat/L) with a mean of 1,504.5 ± 297.5 U/L (25.1 ± 5.0 µkat/L). Fifty-nine patients had chest compressions performed, with mean number of chest compressions 521.7 ± 54.3. The four patients who did not receive chest compressions were treated with defibrillation or cardioversion and had medications such as epinephrine and atropine administered. For all 59 patients who had chest compressions performed, the number of chest compressions showed a linear association with post-CPR CK as well as post-CPR logCK levels (post-CPR CK, r = 0.498, p <0.001; post-CPR logCK, r = 0.535, p <0.001). For patients receiving electrical counter-shock or cardioversion, the number of joules administered ranged from 200 to 3,300 with a mean of 646.1 ± 122.1 J and also showed a linear association with post-CPR CK levels (r = 0.742, p <0.001). Post-CPR CK-MB levels for patients in which this was positive ranged from 15.2 to 1,309.4 U/L (0.25 to 21.8 µkat/L) with a mean of 399.1 ± 96.3 U/L (6.7 ± 1.6}
\[ \text{\(\mu\text{kat/L}\)} \text{ and also showed a positive correlation with the number of joules administered (r = 0.655, p<0.005), the number of joules for these patients ranging from 200 to 3,300 J with a mean of 795.3 \pm 187.3 \text{ J. For patients receiving chest compressions and with positive post-CPR CK-MB fraction, the number of chest compressions ranged from 200 to 1,800 with a mean of 676.8 \pm 93.2, and this also showed a positive correlation (r = 0.474, p<0.02) with post-CPR CK-MB levels (range 15.2 to 1,309.4 U/L [0.25 to 21.8 \mu\text{kat/L}], mean 322.6 \pm 77.0 \text{ U/L [5.4 \pm 1.3 \mu\text{kat/L}]}). Seven (11.1 percent) of the 63 patients were hypokalemic postcardiac arrest (serum potassium <3.5 mmol/L), 47 patients (74.6 percent) were normokalemic (serum potassium between 3.5 and 5.3 mmol/L inclusive), and the nine remaining patients (14.3 percent) were hyperkalemic (serum potassium >5.3 mmol/L). There was no linear association between the CK values and serum potassium concentrations. Post-CPR phosphate levels were available for only 47 patients. Of these, nine (19.1 percent) were hypophosphatemic (serum phosphate <2.7 mg/dl [0.87 mmol/L]) during the postcardiac arrest period, 27 patients (57.4 percent) were normophosphatemic (serum phosphate between 2.7 and 4.5 mg/dl [0.87 to 1.45 mmol/L] inclusive), and the 11 remaining patients (23.4 percent) were hyperphosphatemic (serum phosphate greater than 4.5 mg/dl [1.45 mmol/L]). There was no correlation between the serum concentrations of phosphate and serum CK values. Forty-three (68.3 percent) of the 63 patients had postcardiac arrest CK levels greater than 224 U/L (3.7 \mu\text{kat/L}) and were included in group 1. Forty of these patients had chest compressions performed (the other three had only defibrillation and medications administered) and for these patients the mean number of chest compressions was 759.5 \pm 87.4 while the mean CK was 2,140.2 \pm 400.4 \text{ IU/L (35.7 \pm 6.7 \mu\text{kat/L})}. Laboratory data of group 1 and group 2 patients is shown in Table 1. There was no difference in concentrations of serum potassium between patients with and without rhabdomyolysis. Similarly, there was also no difference between the serum levels of phosphate in the patients of group 1 vs group 2. We further subdivided group 1 into patients who had chest compressions alone and patients who had both chest compressions and defibrillation performed. Twenty patients received chest compressions alone and had a mean CK value of 1,399 \pm 310 \text{ IU/L (23.3 \pm 5.2 \mu\text{kat/L})}. There were 20 patients who had both chest compressions and defibrillation and this group had a mean CK value of 2,948 \pm 701 \text{ IU/L (49.1 \pm 11.7 \mu\text{kat/L})}. Serum CK levels were higher (p<0.05) in the patients who received chest compression as well as electric countershock therapy when compared with the patients who received chest compressions alone.

Table 1—Laboratory Data of Patients with (Group 1) and without (Group 2) Rhabdomyolysis*

<table>
<thead>
<tr>
<th>Variable (Serum)</th>
<th>Group 1 (n = 43)</th>
<th>Group 2 (n = 20)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium, mmol/L</td>
<td>138.0 \pm 1.0</td>
<td>137.0 \pm 1.0</td>
<td>&lt;0.2</td>
</tr>
<tr>
<td>Potassium, mmol/L</td>
<td>4.4 \pm 0.1</td>
<td>4.4 \pm 0.3</td>
<td>&gt;1.0</td>
</tr>
<tr>
<td>Phosphate, mmol/L</td>
<td>1.2 \pm 0.13</td>
<td>1.7 \pm 0.16</td>
<td>&lt;0.2</td>
</tr>
<tr>
<td>Bicarbonate, mmol/L</td>
<td>20.0 \pm 1.0</td>
<td>20.0 \pm 1.0</td>
<td>&lt;0.2</td>
</tr>
<tr>
<td>Glucose, mmol/L</td>
<td>12.7 \pm 1.4</td>
<td>12.2 \pm 1.2</td>
<td>&lt;0.2</td>
</tr>
<tr>
<td>LDH, \mu\text{kat/L}</td>
<td>26.5 \pm 6.9</td>
<td>10.4 \pm 4.3</td>
<td>&lt;0.2</td>
</tr>
<tr>
<td>CK, \mu\text{kat/L}</td>
<td>38.0 \pm 6.4</td>
<td>1.6 \pm 0.2</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

*LDH = lactic dehydrogenase; CK = creatine kinase.

For those 20 patients who had postcardiac arrest CK levels less than 224 U/L (3.7 \mu\text{kat/L}), or group 2 patients, we calculated a ratio of peak post-CPR CK activity with baseline activity and for 19 of these patients (one had a baseline CK level of 0 and thus a ratio was not calculated), the ratio ranged from 1.37 to 5.95 (mean ratio = 2.9). Eight of these 20 patients were overtly cachectic either because of systemic malignancy or malnutrition. Another ten patients also showed hypalbuminemia (mean, 2.3 g/dl [23 g/L]; range, 1.5 to 3.3 g/dl [15 to 33 g/L]). This suggests that those patients without rhabdomyolysis did show increases in CK levels but did not fit into the established criteria of rhabdomyolysis because these patients had a lower lean body mass.

There were 30 patients with known CAD (as defined in the "Methods" section) and 33 patients without (Table 2). Of those 30 patients with known CAD, 18 (60 percent) had CK-MB present and of the 33 patients without CAD, ten (30.3 percent) had CK-MB present postcardiac arrest (Table 2). To determine the level of significance of these proportions, we applied the \( \chi^2 \) test and obtained a value of 4.55 which for 1 df has \( p<0.05 \).

For patients without CAD, we compared CK levels, number of chest compressions, and number of joules given for patients with positive vs negative CK-MB fraction (Table 3). The patients with positive CK-MB fraction had significantly higher total CK levels (all but one patient had levels greater than 1,900 U/L [31.7 \mu\text{kat/L}]) (p<0.001), number of chest compressions given (p<0.01), and cumulative number of joules given during defibrillation (p<0.001) compared with patients with negative CK-MB fraction (Table 3, Fig 1

Table 2—CK-MB Fraction in Patients with and without Coronary Artery Disease (CAD)*

<table>
<thead>
<tr>
<th>With CAD</th>
<th>Without CAD</th>
<th>Total Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive CK-MB</td>
<td>18</td>
<td>10</td>
</tr>
<tr>
<td>Negative CK-MB</td>
<td>12</td>
<td>23</td>
</tr>
<tr>
<td>Total patients</td>
<td>30</td>
<td>33</td>
</tr>
</tbody>
</table>

*CK-MB = MB fraction of CK enzyme.
Table 3 — Comparison of Creatine Kinase (CK) Levels, Chest Compressions (CC), and Joules (J) Administered for Patients with and without CAD and with Positive or Negative CK-MB Fraction*

<table>
<thead>
<tr>
<th>(-)CAD</th>
<th>(+)CAD</th>
</tr>
</thead>
<tbody>
<tr>
<td>(+)MB</td>
<td>(-)MB</td>
</tr>
<tr>
<td>CK</td>
<td>46.6 ± 12.5</td>
</tr>
<tr>
<td>CC</td>
<td>392 ± 126</td>
</tr>
<tr>
<td>J</td>
<td>1,025 ± 71</td>
</tr>
</tbody>
</table>

* (-)CAD = no known coronary artery disease (CAD); (+)CAD = known coronary artery disease; (-)MB = negative CK-MB fraction; (+)MB = positive CK-MB fraction; CK = mean total creatine kinase (µkat/L); CC = mean number of chest compressions; J = mean cumulative number of Joules.

through 3). For patients with known CAD there were no significant differences in CK levels, chest compressions, or number of Joules between patients with positive and negative CK-MB fraction (Table 3, Fig 1 through 3).

Mean serum post-CPR CK levels were higher for patients who received larger doses of intravenous epinephrine but this relationship was not statistically significant due to large standard error. No relationship was found between mean CK levels and administration of other medications such as atropine, sodium bicarbonate, calcium chloride, dextrose, or lidocaine.

Clinical and biochemical profiles in patients surviving CPR without neurologic sequelae (group A, n = 20) and in patients who died in the hospital (n = 41) or who were discharged but with severe neurologic impairment (n = 2) (group B, n = 43) are shown in Table 4 and in Figure 4. Mean age was 63.2 ± 2.9 years (range, 34 to 84 years) in group A patients vs 66.7 ± 2.5 years (range, 24 to 95 years) in group B patients (p<0.2). Of group A patients, two (10.0 percent) were hypokalemic (serum potassium level <3.5 mmol/L), 16 were normokalemic (serum potassium level between 3.5 and 5.3 mmol/L inclusive), and two (10.0 percent) were hyperkalemic (serum potassium level >5.3 mmol/L) with a mean of 4.3 ± 0.2 (range, 2.9 to 5.6 mmol/L). Five patients (11.6 percent) in group B were hypokalemic, 31 were normokalemic, and seven (16.3 percent) were hyperkalemic as defined above with a mean potassium level of 4.5 ± 0.2 mmol/L (range, 3.1 to 7.5 mmol/L). Mean serum potassium level did not differ significantly between groups A and B (p<0.2). Post-CPR phosphate values were available for only 16 group A patients. Five (31.3 percent) of these patients were hypophosphatemic (serum phos-
Table 4—Comparison of Clinical and Biochemical Parameters between Patients Surviving CPR without (group A) and with (group B) Significant Long-term Neurologic Sequelae or Death before Discharge from the Hospital*  

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group A (n=20)</th>
<th>Group B (n=43)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>63.2±2.9</td>
<td>66.7±2.5</td>
<td>&lt;0.2</td>
</tr>
<tr>
<td>Duration CPR, min</td>
<td>4.7±0.7</td>
<td>9.5±1.1</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Potassium, mmol/L</td>
<td>4.3±0.2</td>
<td>4.5±0.2</td>
<td>&lt;0.2</td>
</tr>
<tr>
<td>Phosphate, mmol/L</td>
<td>1.1±0.1</td>
<td>1.4±0.1</td>
<td>&lt;0.1</td>
</tr>
<tr>
<td>Creatinine, μmol/L</td>
<td>156±40</td>
<td>241±40</td>
<td>&lt;0.2</td>
</tr>
<tr>
<td>Creatine kinase, μkat/L</td>
<td>25.8±6.9</td>
<td>26.2±6.4</td>
<td>&lt;0.2</td>
</tr>
</tbody>
</table>

* CPR = cardiopulmonary resuscitation.

Phosphate level <2.7 mg/dl [0.87 mmol/L], nine were normophosphatemic (serum phosphate level between 2.7 and 4.5 mg/dl [0.87 and 1.45 mmol/L] inclusive), and two (12.5%) were hyperphosphatemic (serum phosphate level >4.5 mg/dl [1.45 mmol/L]) with mean 4.5±0.4 mg/dl (1.1±0.1 mmol/L) and range 1.3 to 6.9 mg/dl (0.4 to 2.2 mmol/L). For group B patients, 31 had post-CPR phosphate levels available. Of these, four (12.9%) were hypophosphatemic, 18 were normophosphatemic, and nine (29.0%) were hyperphosphatemic as defined above with mean 4.5±0.4 mg/dl (1.4±0.1 mmol/L) and range 1.4 to 11.3 mg/dl (0.5 to 3.6 mmol/L) for the entire group. The serum phosphate difference between the two groups had a p value of <0.1. For group A patients mean CPR duration was significantly less compared with group B patients (4.7±0.7 min; range, 2 to 15 min for group A and 9.5±1.1 min, range 2 to 30 min for group B patients, p<0.01). Five group A patients (25.0%) who had elevated serum creatinine (>1.4 mg/dl [124 μmol/L]) while in the remainder it was normal (mean group A 1.8±0.5 mg/dl [156±0.4 μmol/L], range 0.5 to 8.8 mg/dl [44.2 to 778 μmol/L]). For group B patients, 24 (55.8%) had elevated serum creatinine with overall mean 2.7±0.5 mg/dl (241±40 μmol/L), range 0.6 to 14 mg/dl (53 to 1,238 μmol/L) and a p value of <0.2 between groups A and B. The CK values ranged from 52 to 6,143 U/L (0.87 to 102.4 μkat/L) with a mean of 1,547±415 U/L (25.8±6.9 μkat/L) for group A patients and for group B CK values ranged from 17 to 8,750 U/L (0.3 to 145.8 μkat/L) with a mean of 1,572±383 U/L (26.2±6.4 μkat/L). The CK values did not differ significantly between the two groups. Eight (40.0%) of the group A patients had a positive CK MB-fraction post-CPR while 18 (41.8%) of the group B patients had a positive CK MB-fraction (data not shown).

**Discussion**

The present study demonstrates an association between the number of chest compressions administered during resuscitative efforts after cardiac arrest and post-CPR CK levels. It also shows a direct relationship between the cumulative number of joules administered via defibrillation and post-CPR CK levels as well thereby indicating that physical injury caused by chest compressions and electrical burn injury from defibrillation are the important determinants of muscle cell injury in CPR. A significant number of these patients (43 [68.3%] of 63) developed rhabdomyolysis. In patients with rhabdomyolysis, those who had received both chest compressions and defibrillation had significantly higher (p<0.05) postcardiac arrest CK values compared with patients given chest compressions alone showing that both physical and electrical injury contributed to rhabdomyolysis. It is noteworthy that of the 20 patients who did not fit into the conventional biochemical definition of rhabdomyolysis (serum CK levels over 1.5 times of upper limit of normal values), all of them had increases compared with their baseline levels indicating that perhaps they did also suffer some degree of muscle cell injury. Serum CK levels have been shown to correlate directly with lean body mass. Thus, low baseline levels of CK in many of the patients of group 2 or without rhabdomyolysis were suggestive of smaller lean body mass in these patients. This was further supported by the finding that eight patients in group 2 were overtly cachectic and another ten had hypalbuminemia. Another explanation, though less likely, is variability in force of compressions among CPR personnel (some individuals may compress with greater force than others) and variability in conductivity between defibrillation paddles and the skin (insufficient electrode gel might diminish the amount of electrical energy imparted to the chest). In animal experiments and in human situations, the occasional transient increases in serum muscle en-

![Figure 4](http://journal.publications.chestnet.org/pdaccess.ashx?url=data/journals/chest/21644/ on 06/26/2017)
zyme levels following direct current countershock cardioversion have been attributed to chest wall muscles and not to cardiac muscle damage.\textsuperscript{6,12} In an isolated study, the effect of cardioversion on serum CK showed that MB fraction of CK was elevated in a small number of patients.\textsuperscript{12} However, none of these studies included patients who received chest compressions as well as direct current countershock therapy. Thus, the present study, in which we observed elevation of CK-MB fraction in almost 50 percent of the patients, is not comparable to other studies. Also, for patients with a positive CK-MB fraction, the level of CK-MB was significantly correlated with the number of joules administered, thereby suggesting that electrical injury had played a role in the elevation of CK-MB fraction in these patients.

Both hypokalemia and hypophosphatemia are well-known causes of rhabdomyolysis.\textsuperscript{14} Recently, we have also reported that 28 percent of hypokalemic patients had elevated serum CK levels.\textsuperscript{5} In the present study, seven patients were hypokalemic and nine patients were hypophosphatemic. Thus, it seems that hypokalemia or hypophosphatemia might have contributed to the development of muscle cell injury in some of these patients. However, we were unable to show any direct relationship between muscle cell injury and serum concentrations of these electrolytes. It is possible that true deficiency of these electrolytes was masked because of ongoing rhabdomyolysis with consequent release of potassium and phosphate.

Our data also showed a much greater (p<0.05) likelihood of a patient with known CAD (as defined in the "Methods" section) having the MB fraction of CK present after CPR when compared with patients without a known history of CAD. Sixty percent of patients with a history of CAD had CK-MB present following CPR while only 30.3 percent of patients without known CAD had a positive CK-MB fraction (ten of 33 patients). In addition, our data show that for patients without known CAD, total CK elevation (above 1,900 U/L [31.7 μkat/L]), physical injury, and electrical injury are determinants of CK-MB positivity, whereas for patients with known CAD, there are no significant differences in these parameters between patients with positive and negative CK-MB fraction. These data suggest that CK-MB elevation in patients without known CAD may be secondary to repeated and prolonged direct trauma to the heart from more extensive physical and electrical injury, whereas patients with CAD may develop positive CK-MB fraction with lesser degrees of chest cavity trauma perhaps because of additional ongoing myocardial ischemia secondary to coronary artery stenosis with superimposed hypotension. However, we cannot confirm this speculation in the absence of thallium studies in the post-CPR period.

Our data also show that patients who survive CPR to be discharged from the hospital with intact neurologic status tend to be those with shorter duration of CPR. Possibly a longer duration of CPR results in prolonged ischemia, although another factor may be that patients requiring longer duration of resuscitative efforts might have a poorer underlying general medical condition making resuscitation more difficult and long-term survival less likely. This may be reflected in the fact that the patients without long-term survival following CPR had higher serum creatinine levels, although this did not reach significance in our sample. Serum phosphate levels were somewhat higher in group B patients, although this occurred mostly in patients with elevated creatinine concentrations. Although serum phosphate levels did not differ significantly between group A and B patients, it is of note that a greater percentage of group B patients were hyperphosphatemic (29.0 percent vs 12.5 percent for group A) and a greater percentage of group A patients were hypophosphatemic (29.4 percent vs 12.9 percent for group B). Thus, although the mean phosphate values did not differ significantly, our data show that patients who do not survive to hospital discharge after CPR or who suffer severe neurologic damage are more likely to have an elevated serum phosphate level. A greater percentage of group B patients were hyperkalemic as well (16.3 percent vs 10.0 percent for group A patients). It is noteworthy also that although mean serum creatinine levels did not significantly differ between groups A and B, group B patients were much more likely to have an elevated serum creatinine level (55.8 percent vs 25.0 percent for group A). The higher proportion of group B patients with elevated potassium and phosphate levels may be related to the increased likelihood for group B patients to have an elevated serum creatinine level. These data suggest that the presence of renal insufficiency may be an indicator of an adverse long-term outcome following CPR. Group B patients were no more likely to have a positive CK-MB-fraction in our sample.

We conclude that the majority of patients show an elevation of CK levels in the post-CPR period. The CK levels show a direct relationship with physical injury resulting from the number of chest compressions and electrical injury imposed by the amount of electric current used for countershock therapy. CK-MB levels also appear to be related to the electrical and physical injury sustained during CPR and are significantly more likely to be found in patients with CAD. Our data also suggest that the presence of CK-MB after CPR may serve only as a crude indicator of underlying CAD and elevated CK-MB fraction may be a consequence of direct physical and electrical injury rather than the effects of ischemic cardiac events in patients with no known CAD.
 Patients who are successfully resuscitated but who suffer severe neurologic damage or who later die in the hospital tend to be those who had longer duration of CPR and are more likely to have elevated serum potassium, phosphate, and creatinine values. The presence of renal insufficiency may have an adverse effect on the long-term outcome of CPR.

REFERENCES
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