Creatine Kinase and Lactate Dehydrogenase Isoenzymes in Serum of Patients Suffering Burns, Blunt Trauma, or Myocardial Infarction

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Medical records of 53 burn and trauma patients were reviewed to assess the possibility of myocardial damage. Except for electrophotically detectable creatine kinase MB isoenzyme, none showed evidence of myocardial injury. Lactate dehydrogenase isoenzyme tests, electrocardiograms, myocardial pyrophosphate scans, clinical course, and results of two autopsies were all negative for myocardial necrosis or ischemia. Types of patient, number, mean peak value (U/L) for serum creatine kinase and ranges of percent age MB isoenzyme were as follows. Burns from direct electrical contact: 28, 16, 600, 0–29; electrical flash or other thermal burns: 10, 4340, 0–22; blunt trauma (mostly from automobile accidents): 15, 3430, 0–18; myocardial infarction: 57, 1520, 4–46. Evidently creatine kinase MB isoenzyme is nonspecific in burn and trauma patients and should not be the only test result used to assess myocardial involvement.

Additional Keyphrases: heart disease • "flipped" LD-1/LD-2 ratio • test sensitivity • test specificity • enzyme activity

Creatine kinase (CK, EC 2.7.3.2) is a dimeric protein. The two types of subunits (M and B) give rise to three isoenzymes: creatine kinase MM isoenzyme (CK-MM), MB isoenzyme (CK-MB), and BB isoenzyme (CK-BB). Myocardium contains CK-MB, which constitutes 15 to 50% of total CK activity in this tissue (1–6). The only other reported major source of CK-MB is skeletal muscle; however, the consensus has been that this tissue contains only 1 to 4% of total CK as CK-MB (2, 6–9). In fact, the demonstration of abnormally high activities of serum CK-MB is claimed to be a specific and extremely sensitive index of myocardial necrosis or ischemia (1, 9–11). Several investigations report little or no CK-MB in skeletal muscle (1, 3, 12–15), but some (4, 16, 17) find as much as 20 to 30% of total CK in skeletal muscle to be CK-MB. In cases of skeletal muscle trauma without myocardial injury <2–8% (5, 9, 18–20), and in one case of crush injury >10% (16) of total CK in serum was CK-MB.

Lactate dehydrogenase (LD, EC 1.1.1.27) is a tetrameric protein. The four subunits give rise to five isoenzymes, designated LD-1 through LD-5. This enzyme is abundant in almost all tissues; consequently, total LD assay is a nonspecific test. However, specificity is greatly increased by analysis of the isoenzyme pattern along with total LD. Heart, kidney, brain, and erythrocytes have the highest proportions of LD-1 and LD-2 (65–75% of the total LD); liver and skeletal muscle have the highest proportion of LD-5 (60–70% of the total LD) (21). LD-1 concentration (22–24), LD-1/total LD ratio (%LD-1) (24–27), and LD-1/LD-2 ratio (9, 11, 28, 29) are among the tests recommended for laboratory diagnosis of myocardial infarction (MI). The best predictive test for diagnosis of MI is the combined serial examination of CK and LD isoenzyme patterns (23, 30).

There is increasing controversy about the usefulness of CK-MB analyses in assessment of myocardial involvement in trauma (5, 9, 16, 18–20) and, in our experience, in patients with electrical and thermal injuries. In this paper we compare the enzyme and isoenzyme patterns of 38 burn and 15 trauma patients without apparent myocardial injury with those of 57 patients with clinically-confirmed MI.

Materials and Methods

Patients

This study involved 110 patients admitted to the University of Utah Hospital between June 1981 through November 1983. We included all patients suffering from burns, trauma, or clinically-confirmed MI for whom isoenzyme determinations were performed between July 1982 through November 1983. Six known cases of electrical injury between June 1981 and November 1981 were also included.

Medical records of all burn and trauma patients were reviewed to check for any clinical evidence of myocardial injury. Table 1 lists the results of each test performed for the burn and trauma patients to assess myocardial involvement. For each patient, electrocardiograms were reviewed for diagnostic QRS-wave abnormalities and the hospital course was evaluated for evidence of ventricular dysfunction or cardiac arrhythmias, independent of CK or LD isoenzyme results.

Twenty-eight electrical-burn patients sustained a mean of 9% (range, <1–32%) total body surface area burns, of which a mean of 4% (range, <1–20%) of the area was third-degree burn as a result of direct contact with a mean of 18 000 V (range, 140–72 000 V). Electrical entrance wounds were located in the head, neck, or upper extremity in 16 of these patients, the trunk in one, and the lower extremity in two. There was no entrance wound in the remaining nine patients.

The thermal injury group included six electrical flash-burn victims who had experienced burns over a mean of 18%.

| Table 1. Tests Used in Assessing Myocardial Involvement in Burn and Trauma Patients |
|----------------|----------------|----------------|
| isoenzyme      | No. patients tested |
| CK             | Serial ECGs*    | Myocardial pyrophosphate scans | Autopoles |
| LD             |                 |                             |            |
| 28             | 21              | 28                           | 24         |
| 10             | 6               | 9                            | 4          |
| 15             | 11              | 15                           | 3          |

*Electrocardiograms.
(range, 8–42%) of total body surface area, with a mean of 9% (range, <1–42%) of the area being third-degree burns. Four patients suffered burns over a mean of 27% (range, 10–47%) of body area, with a mean of 17% (range, <1–38%) being third-degree burns from non-electrical sources.

The 15 blunt-trauma patients had had motor vehicle accidents (13 patients) or falls (two patients). Most patients suffered multiple fractures, one patient had facial lacerations as a result of a fall resulting from ethanol intoxication, and another had severe crush injury of a lower extremity.

Histories were recorded for all the burn and trauma patients (49 males and four females, age range 6 to 79 years) with respect to cardiac arrest and arrhythmias.

In our study of sensitivity and specificity of the isoenzyme tests for diagnosis of MI, we included all patients between July 1982 through November 1983 with isoenzyme results at the University of Utah Hospital. All of these patients were checked for any clinical evidence of myocardial necrosis or ischemia. All of the 57 clinically-confirmed MI patients included in this study were admitted during the above time period, and all were included in the sensitivity and specificity studies.

Methods and Instrumentation

Total serum CK activity was measured spectrophotometrically in the acc II discrete analyzer (Clinical Systems Division, Du Pont Co., Wilmington, DE 19889). Total serum LD activity was measured in either the acc II or the smac continuous-flow analyzer (Technicon Instruments Corp., Tarrytown, NY 10591). CK and LD isoenzymes were separated electrophoretically on agarose gels (Corning Medical and Scientific Works, Medfield, MA 02052), and quantified with an autoscanning densitometer (Helena Laboratories, Beaumont, TX 77074). The LD-1 activities for each patient were determined from total LD activities and the %LD-1 given by isoenzyme electrophoresis.

Initial electrocardiograms were recorded for all but one patient (Table 1) within the first 20 to 36 h after admission, with standard 12-lead machines, and such recordings were made daily for at least three days.

Pyrophosphate scans were performed as follows. Patients were injected with 15 to 25 mCi of Tc-99m stannous pyrophosphate 24 to 36 h after injury and total body images were recorded 2–4 h after the injection with a mobile scintillation camera having a low-energy parallel-hole collimator (Picker International, Inc., Northford, CT 06472). In all cases, anterior, left anterior oblique, and left lateral views were obtained over the heart.

To evaluate for cardiac arrhythmias, all patients were evaluated by bedside monitoring or 24-h Holter recordings, started within 12 h of injury. In no case was cardiac arrhythmia a significant problem requiring therapy.

Number of Isoenzyme Determinations

The number of separate CK isoenzyme determinations for each subject ranged from one to 10 (mean, 2.9) for patients suffering electrical injuries; from one to five (mean, 2.8) for those who had sustained thermal injuries; from one to seven (mean 2.9) for trauma cases; and from one to seven (mean, 3.4) for MI patients. The number of separate LD isoenzyme analyses was one to seven (mean, 2.4) for patients who had experienced electrical injuries, one to five (mean, 3.0) for those suffering thermal injuries, one to three (mean, 2.0) for trauma patients, and one to seven (mean, 3.3) for MI cases.

Reference Intervals

The normal reference interval for total CK in our labora-

tory is 36–188 U/L at 37 °C, with 0% CK-MB. For total LD it is 138–328 U/L at 37 °C, with 21.5–31.1% LD-1 and 2.5–12.5% LD-5.

Statistics

Student's t-test was used to compare isoenzyme values between patient groups. We used linear regression analyses in correlating total CK activity with %CK-MB in different patient categories. We computed the standard error of the slope of the regression line for each group of patients, to determine the level of significance at which each slope differed from zero.

Results

Creatine Kinase

CK and %CK-MB. We serially measured total CK activity and %CK-MB in all patient categories (Figure 1). As expected, all patients with clinically confirmed diagnosis of MI had increased CK-MB activity, to at least 4% of total CK activity. However, we also detected at least 2% CK-MB in 16 of the 28 electrical burn patients, four of the 10 thermal burn patients, and 10 of the 15 trauma patients. One trauma case was notably different; a severe crush injury of a lower extremity gave rise to a peak CK of 35 400 U/L and a peak value of 18% for CK-MB. There was no significant correlation between the extent of increase in CK and the site and extent of muscle damage caused by the injury (and the consequent surgery), as reflected by pyrophosphate scans of the burn and trauma patients.

The highest ratios of peak CK to peak %CK-MB (Figure 2) were for electrical burn patients (610–10 000 U/L). In contrast, for MI patients this ratio ranged from 10 to 950 U/L.

A correlation coefficient of 0.62 was found between peak CK activity and peak %CK-MB in electrical-burn patients (Figure 3). Except for two outliers, all burn, trauma, and MI patients were included in the analyses. Only for electrical-burn patients was the regression line slope significantly different from zero (p<0.001).

CK-MB specificity and sensitivity in diagnosis of MI. Between July 1982 through November 1983 we measured CK isoenzymes in serum specimens from 634 patients, of whom 118 had above-normal (≥2%) proportions of CK-MB.

Fig. 1. Total CK and %CK-MB

Distribution and mean of peak CK activity ( ) and peak %CK-MB ( ) values in burn, trauma, and MI patients. Peak CK activity is plotted on a logarithmic scale. In Figures 1 and 2, the points shown in parentheses were more than three standard deviations above the mean, and were not included in the calculation of the mean values. The numbers by the horizontal line segments are the mean values. The horizontal line segments above (and in one case also below) the lines representing the means correspond to mean ± 2 standard deviations.
There were 57 cases of MI, as diagnosed by electrocardiographic changes and clinical course in addition to isoenzyme patterns. All patients with chest pain and diagnostic electrocardiographic changes had above-normal (≥4%) CK-MB activities, yielding a CK-MB sensitivity of 100% in diagnosis of MI. The remaining 61 patients with increased CK-MB included 11 electrical burn, 10 trauma, and three thermal burn patients. Calculated on the basis of these data, the specificity of CK-MB in diagnosis of MI in our patient population is 89%.

Lactate Dehydrogenase

LD and %LD-5. Total LD activities were about equally increased in each group. The means were 1.8 to 2.7 times greater than the upper reference limit, 328 U/L (Figure 4). However, the proportion of LD-5 was generally greater in burn (mean, 31 and 32%) and trauma (mean, 22%) patients than in MI cases (mean, 15%).

LD-1, %LD-1, and LD-1/LD-2 ratio. The mean LD-1 activity and %LD-1 for the MI patients were significantly greater (p < 0.05) than in the burn and trauma patients (Figure 5). The mean %LD-1 (15–16%) was below the lower limit of the normal reference interval (21.5%) in the burn and trauma victims, but for the MI patients it was 39%, which is above the upper normal reference interval (31.1%). The mean LD-1/LD-2 ratio for the MI patients (1.26) exceeded normal reference intervals (0.45–0.83) for this variable (28, 31), but this ratio for our burn and trauma patients (0.63–0.69) was well within the normal reference interval (Figure 6). This ratio for the MI patients was significantly greater (p < 0.001) than for the burn and trauma patients.

Specificity and sensitivity of LD-1/LD-2 ratio in diagnosis of MI. Between July 1982 through November 1983 we analyzed serum specimens from 512 patients for LD isoenzymes, of whom 47 had LD-1 activity > LD-2 activity ("flipped" LD). Among the latter group, 41 had MI as diagnosed from clinical symptoms and serial electrocardiograms in addition to supranormal results for serum CK-MB and CK activities. None of the remaining six patients with flipped LD had detectable CK-MB activity in their serum, and none was suspected of having MI. From these data we calculate a specificity of 99% for LD flip and 100% for the combined CK and LD isoenzyme analyses. During this period, there were 54 MI patients for whom we had LD isoenzyme results. Thirteen had no LD flip; thus the sensitivity was only 76% for the LD flip in diagnosis of MI.

Assessment of Myocardial Damage in Burn and Trauma Patients

Except for increased CK-MB in some burn and trauma patients (Figure 1), there was no other evidence for myocardial ischemia or necrosis. Table 1 lists the number of different tests performed in each group of patients. Clinical observations, serial LD isoenzyme determinations, serial electrocardiograms, Tc-99m sestamibi pyrophosphate myocardial scans, and results of two autopsies all suggest the absence of myocardial injury or necrosis. Eighteen patients demonstrated nonspecific ST-T wave abnormalities, two had prolonged QT intervals, and three showed partly flattened T waves. None of these abnormalities was considered diagnostic of myocardial injury. In one patient, Holter monitoring demonstrated a transient Wenckebach phenomenon, which
cleared during the course of the monitoring period. Results of all other Holter recordings were within normal limits. Six of the burn and trauma patients had cardiac arrests, four of them fatal. One electrical-burn patient developed ventricular arrhythmia of unknown etiology towards the end of his hospitalization and died from it after an episode of cardiac arrest.

Time between Injury and Peak CK, CK-MB, and LD Activities in Burn and Trauma Patients

The intervals after injury until peak CK, CK-MB, and LD activities were compared for the burn and trauma patients and the MI patients (Table 2). We included in the analysis only patients with more than one isoenzyme determination and with at least one specimen containing detectable CK-MB. The average intervals between injury and peak CK and CK-MB activities for each patient group were somewhat similar (Table 2), but in some electrical-burn and trauma patients peak CK and (and CK-MB) activities were measured earlier than in MI patients, e.g., 4–5 h vs 12–24 h in MI patients (11, 14, 32, 33). Peak LD activities also appeared, on the average, earlier after electrical burn and trauma, 14–18 h as compared with 48 h for MI (32). True peak enzyme activities were approximated in each group of burn and trauma patients, as is apparent from the following analysis. The average time period between injury and the last isoenzyme analysis, \( t_w \), was determined for each group, as were two other parameters: the average time interval for successive isoenzyme analyses, \( t_i \), and the average time between injury and maximum enzyme activities, \( t_m \). The \( t_w/t_m \) ratio was 2.8–4.5 for electrical burn, 1.2–1.7 for thermal burn, and 1.6–2.5 for trauma patients. Therefore, the observation periods for enzyme activities generally encompassed the time periods between injury and peak enzyme activities. The \( t_w/t_m \) ratio was only 0.4 to 1.1 for the burn and trauma patients. Therefore, the serial isoenzyme measurements were frequent enough to approximate the time between injury and peak enzyme activities for each group of patients.

Discussion

Specificity and Sensitivity of the Isoenzyme Tests in Diagnosis of MI

CK-MB analysis is claimed to be the single most sensitive

Table 2. Time between Injury or Onset of Chest Pain and Peak CK, CK-MB, and LD Activities in Burn, Trauma, and MI Patients

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<th>CK</th>
<th>CK-MB</th>
<th>LD</th>
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<td></td>
<td>Hours after injury or onset of chest pain</td>
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<td>4.0–35</td>
<td>4.0–30</td>
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<td>(17)</td>
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<td>15–34</td>
<td>11–30</td>
<td>9.0–67</td>
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*The numbers in parentheses below each range are the arithmetic means corresponding to each range. The average number of CK isoenzyme analyses was 3.6 for electrical burn, 2.7 for thermal burn, and 3.8 for trauma patients. The average number of LD activity measurements was 2.9 for electrical burn, 3.5 for thermal burn, and 2.4 for trauma patients. * References.
and specific index to MI (1, 9–11, 18). However, such reports generally suffer from a population bias because subjects were selected from coronary-care units, where the prevalence of acute MI is approximately 50% (34). The specificity of the CK-MB assay is considerably less when it is used in populations other than patients in coronary-care units (35). CK-MB elevation has been documented in certain myopathies (36), Duchenne muscular dystrophy (7), profound hypothermia (37), malignant hyperpyrexia (38), polymyositis–dermatomyositis (4, 8, 39), Reye’s syndrome (40), newborns, infants, and children (41), trauma to skeletal muscle (5, 9, 16, 18–20), and upon vigorous or sustained physical exercise (42–46). The origin of most CK-MB under these conditions is skeletal muscle (45–47). In view of this array of non-MI conditions in which serum CK-MB activity is increased, it is not surprising that we obtained a CK-MB specificity of only 89% in our sample, which included burn and trauma victims in addition to MI patients; the reported average electrophoretic CK-MB specificity for diagnosis of MI is 97% in patients in coronary-care units (9). Diagnosis of MI should never be based on the presence of CK-MB in serum, but should be supported by additional clinical and laboratory data (49).

Flipped LD was 99% specific for diagnosis of MI. In addition to myocardial ischemia or necrosis, an LD-1/LD-2 ratio <1 has been found in hemolytic (49) associated with less hemolytic activity, hemolytic anemia, purpura, disseminated intravascular coagulation, acute renal-cortex infarction (21, 50), muscle injury to type 2 fibers in Duchenne muscular dystrophy or massive muscle necrosis (rhabdomyolysis) (50), and after long-distance running (51–52). However, upon examination of the LD isoenzyme patterns of 512 patients, we found only six with flipped LD but no other evidence for MI.

In diagnosis of MI, sensitivities of the CK-MB (100%) and the LD flip (76%) were similar to the average reported values of 98% and 75% (9). However, the isoenzyme results are weighted heavily in the clinical diagnosis of MI, so these sensitivities may be underestimated. In fact, patients with microinfarctions may not be so diagnosed because of negative isoenzyme results. Since there is no single “gold standard” for diagnosing or ruling out MI, all results for sensitivity and specificity should be considered only approximations. On the other hand, the sensitivity of LD-1/LD-2 ratio may be underestimated. Frequently, LD flip is not observed when LD isoenzyme analysis is not performed long enough after the onset of chest pain; a CK-MB result along with other findings may have established or ruled out MI without the need for further LD isoenzyme analyses.

None of the six non-MI patients with LD flip had increased CK-MB. If a positive result is interpreted as both detectable CK-MB activity and LD flip, this test combination had a predictive value for a positive result and a specificity of 100% in our study. The combined use of CK and LD isoenzyme analyses reportedly has the best predictive value in diagnosis of MI (23, 30).

Of our burn and trauma patients, 57% had both increased CK-MB activity and increased total CK activity in their serum, 30% having >10% CK-MB. Four of the 15 trauma patients (27%) also had >10% CK-MB: a case of severe crush injury of a lower extremity, two cases of automobile accidents, and a case of free fall. Only one case of crush injury to the lower extremities has been reported in which >10% CK-MB was observed (16). In contrast to our findings, most reports conclude that CK-MB is ≤5% after trauma to skeletal muscle (5, 9, 18–20). Total CK, but not CK-MB, was reported to be increased after intramuscular injections and various surgical procedures (12, 13, 15). However, a transient increase in CK-MB in serum after injury to drivers in automobile accidents (53) was considered nonspecific rather than a reflection of myocardial damage (54). Aside from increased CK-MB, the trauma and burn patients in our study had no other evidence for myocardial involvement. Our findings are in contrast to those reporting only small amounts of CK-MB in skeletal muscle (1, 3, 12–15), but agree with those reporting as high as 20 to 40% CK-MB in skeletal muscle (4, 16, 17). However, 43% of our burn and trauma patients showed no detectable increase in CK-MB in serum, despite total CK values as high as 25 200 U/L. Possibly the proportion of CK-MB in skeletal muscle varies between different diseases, anatomic locations, and individuals (17). And individuals may differ in the rate of CK-MB release from skeletal muscle, the rate of its clearance from the bloodstream, or both.

Figures 1 and 3 clearly illustrate that total CK activity is, on the average, much more increased in burn and trauma (especially electrical burn) patients than in MI patients (mean of 16 600 U/L in electrical burn, 1520 U/L in MI patients). However, CK-MB averaged 5–9% in burn and trauma patients but 21% in MI patients. Consequently, the ratio of CK to %CK-MB was increased many fold in burn and trauma patients (mean of 1900 U/L in electrical burn and 76 U/L in thermal burn and trauma patients) as compared with MI patients (mean of 70 U/L). There was very little overlap between electrical burn and MI patients when %CK-MB was plotted vs total CK activity (Figure 3). A high ratio of CK to %CK-MB should not be used as the only diagnostic tool to rule out MI. The absence of a CK-MB band on electrophoresis does not exclude myocardial injury, because extensive skeletal muscle damage may mask the effect of lesser myocardial involvement (54). Low CK/%CK-MB ratios (<200 U/L), however, may be indicative of myocardial involvement (Figure 2); none of our burn and trauma patients had CK/%CK-MB ratios of <250 U/L.

LD and LD isoenzymes

Liver disease with congestion, and muscle injury to type I fibers are the two major conditions giving rise to increased LD-5 activity in serum (50). This isoenzyme contributes more to the LD activity in burn and trauma patients (mean of 31–32% in burn and 22% in trauma patients) than in MI patients (mean of 15%) (Figure 4). The mean percentage LD-5 exceeds the upper reference limit (12.5%) in MI. This may be explained by poor hepatic perfusion, which is common in MI patients. The high LD-5 in the burn and trauma patients probably originates from skeletal muscle.

LD-1 activity, %LD-1, and LD-1/LD-2 ratio are all increased in MI patients (9, 22–29). These all averaged higher than the upper reference limits in our MI patients (Figures 5 and 6), and they were distinctly more increased in these patients than in the burn and trauma patients (p < 0.05). LD-1 activity in the burn and trauma patients (70–110 U/L) was increased as compared with the cutoff value of 64 U/L (24), at least partly as a result of total LD elevation (23). But LD-1 in the burn and trauma patients averaged only 15–16% of the total LD, below the lower reference limit (21.5%). This is in part ascribable to the larger contribution of LD-5 to the total LD activity in these patients. The LD-1/LD-2 ratio in burn and trauma averaged only 0.63–0.69, well within reported reference limits (28, 31). Therefore, we suggest that this ratio may be useful to assess myocardial damage in these patients. None of the burn and trauma patients had flipped LD (Figure 6).

Correlation between CK and %CK-MB

CK and %CK-MB were positively correlated (r = 0.62) in electrical-burn patients (p < 0.001 for the regression line
slop being different from zero); generally, patients with highly increased serum CK activity showed detectable CK-MB and a high %CK-MB (Figure 3). However, we found no significant correlation between the extent of muscle injury as reflected by pyrophosphate scans and the degree of increase in CK. Perhaps various muscle tissues have different CK contents and those with relatively high CK content have a relatively high proportion of CK-MB.

Temporal Course of Enzyme Patterns

Except for LD, the temporal course of enzyme increases in burn and trauma patients is quite similar to that in MI patients. CK is present in the cytoplasm and mitochondrion and reaches its maximum in serum earlier in MI than does LD. However, in more severe and sudden muscular traumas such as those caused by electrical and physical injury, LD appears to be released at about the same time as CK, probably as a result of massive muscle cell lysis. Therefore, the LD activity peaks at about the same time as does CK activity (Table 2), because the rate of release is much greater than the rate of decay of these enzymes. LD-5 contributes more to the LD activity in burn and trauma patients than in MI patients (Figure 4). This, and the relatively short biological half-life of LD-5 (4), may also help explain the earlier peak times for total LD in the burn and trauma patients as compared with the MI patients.

In summary, we conclude that:

• CK-MB in our study sample was 100% sensitive, and the combined CK and LD isoenzyme test was 100% specific in diagnosis of MI. Therefore, after detection of serum CK-MB, the LD isoenzyme pattern may establish a positive diagnosis for myocardial injury.

• Low CK/CK-MB ratio (<200 U/L) may be useful in establishing myocardial injury without skeletal muscle involvement; the CK/CK-MB ratio was >250 U/L in all our burn and trauma patients.

• The incidence of myocardial damage as a result of electrical, physical, or thermal trauma seems to be low, based on our examination methods; none of the 53 patients examined had experienced myocardial injury.

• CK-MB is nonspecific in burn and trauma patients and this result alone should not be used to assess myocardial involvement.

• Two negative LD isoenzyme determinations and a negative myocardial pyrophosphate scan, even in the presence of increased CK-MB, appear to rule out significant myocardial injury.

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