Creatine Kinase and Creatine Kinase-MB Isoenzyme during and after Exercise Testing in Normal and Obese Young People*

Alberto Salvadori, M.D.; Paolo Fanari, M.D.; Stefano Ruga, M.D.; Amelia Brunani, M.D.; and Erminio Longhini, M.D.

We report creatine kinase (CK) and CK-MB values during a cycleergometric test up to maximal work capacity in 10 normal subjects aged 20 to 39 years (mean body mass index, 22 kg/m²) and 11 obese patients aged 17 to 42 years (mean body mass index, 41 kg/m²), all without any cardiorespiratory diseases. Total CK was significantly higher in obese patients. The CK-MB was not significantly different between the two groups, except at the first recovery when it was increased in obese patients and decreased in normal subjects. These results could be due to more important total stress of the total musculature, especially cardiac, and especially cardiac musculature in obese patients during a physical effort. Considering the mean values of total CK of our obese patients, it may be possible that they have myocardial damage at percentages of CK-MB less than those of lean subjects generally accepted at more than 4 percent. Moreover, in obese heart patients myocardial distress during exercise testing may be present despite heart rate at peak exercise beneath the theoretic maximal. (Chest 1992; 102:1687-89)

Previous studies of obese subjects at rest have reported an increase in cardiac work, representing percentages of 40 to 190 percent, of more than ideal body weight.1 Necropsy studies on the relationship between heart weight and body weight have demonstrated an increase in heart weight in obese subjects.2 Increments of total creatine kinase (CK) have been noted in the blood of patients with ischemic heart disease when subjected to exercise testing.3,5 Some reports mention a slight elevation in CK-MB isoenzyme after strenuous physical exercise in marathon runners.5,7

Both these increases probably represent a physiologic enzyme leakage from musculature which is paramount to the normal muscular activity and are proportional to the metabolic demands of the muscle.8 The increased body mass of the obese patients requires a greater metabolic energy exchange both at rest and especially during physical exercise.9 To evaluate muscular stress, we recorded CK and CK-MB values during an exercise test up to the maximal work capacity in a group of young obese patients compared with normal young subjects, all without any cardiorespiratory disease.

Methods

We studied 11 obese subjects (5 males) and 10 normal subjects (5 males), whose anthropometric data are reported in Table 1. The body mass index (BMI-kg/m²) was taken as a measure of obesity.10

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<table>
<thead>
<tr>
<th>Table 1 — Anthropometric Data*</th>
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<tr>
<td>Normal subjects</td>
</tr>
<tr>
<td>(range)</td>
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<tr>
<td>No.</td>
</tr>
<tr>
<td>Sex (M/F)</td>
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<tr>
<td>Age (years)</td>
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<tr>
<td>Weight (kg)</td>
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<td>Height (cm)</td>
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<td>BMI (kg/m²)</td>
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Values are mean ± SEM; BMI = body mass index.
†By two tailed analysis of variance.
values differed from those obtained in the control subjects at a
given step of exercise.  

RESULTS

We were not able to differentiate the group of
normal subjects (136 ± 10.6 W [SEM]) by means of
the maximal peak of activity reached. Anaerobic
threshold AT was significantly lower in the obese
subjects (78 vs 110, p < 0.05).

Total CK was significantly higher in the obese
patients at rest, peak of activity, and during first
and late recovery. During exercise, there was no significant
increase in CK in either obese patients or normal
subjects, but the CK of the first recovery in the obese
patients, unlike that in normal subjects, was slightly
higher compared with CK at peak of activity (Fig 1).

The CK-MB was not significantly different in obese
patients and normal subjects at rest or at the peak of
activity and there was no significant increase during
the exercise in either group.

Nevertheless, at the first recovery there was an
increase of the value in obese patients compared with
that at the peak of activity, while there was a decrease
in normal subjects, with a significant difference be-
tween these mean values (Fig 2), which persisted at
the late recovery.

The heart rate was significantly higher in obese
subjects both at rest and when pedaling at zero
resistance; the subsequent increase during exercise
was more pronounced in normal subjects who reached
a significantly higher value at peak activity when
compared with obese patients (Fig 3).

Systolic and diastolic blood pressures increased
during exercise both in normal and obese patients,
with no significant difference between the two groups
(at rest: normal subjects — 116 ± 1.9/77 ± 2.4; obese
patients — 120 ± 2.3/84 ± 2.9; at peak activity: normal
subjects — 153 ± 4.7/90 ± 2.7; obese patients —
155 ± 3.7/95 ± 1.8).

DISCUSSION

The obese subjects show increased total CK values
at rest and during activity and subsequent recovery
when compared with control subjects.

This aspect probably is due to their higher body
mass which is likely characterized by the increase in
muscle mass as well as in fat, and skeletal muscle is
the most important source of the increase in its
enzymatic activity.

No significant difference was noticed at rest in CK-
MB isoenzyme levels between obese and normal
subjects; the slightly increased value in obese patients
could be due to their probable higher heart weight.

At the end of training no significant increase in CK-
MB was measured and this is conceivable since none
of the subjects had myocardial illness. Nevertheless,
at first recovery the obese patients showed a further
increase, while normal subjects showed a slight de-
crease.

From the literature, we know about the very good
correlation between myocardial O₂ consumption and
the product of cardiac frequency with systolic arterial
pressure. By means of this correlation, we can state
that at rest the hearts of obese patients consume more
O₂ than those of control subjects; during exercise,
both groups experience an increase in oxygen con-
sumption at the top of training; and myocardial O₂
consumption in obese subjects is less than that of
normal subjects.

We must remember that cardiac stroke volume
increases to over resting values at low levels of work,
but there is no further increase once cardiac frequency
has risen to 120 beats per minute. At the top of
training both our groups were in this condition.

Obese subjects generally exercise less and are
probably less fit than normal subjects, which means
that their cardiac maximal oxygen consumption may
be reduced.

The behavior of CK-MB in our obese subjects at

Figure 1. Mean CK at rest, at the top of exercise and during
recovery for obese patients compared with normal subjects.

* = p < 0.05; two-tailed analysis of variance and Dunnett method.
At the bottom of the figure the mean values of CK and the
corresponding SEM are given.

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<tr>
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<tr>
<td>CK</td>
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<td>SEM</td>
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<tr>
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<td>119.5</td>
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<tr>
<td>SEM</td>
<td>10.6</td>
<td>11.8</td>
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Figure 2. Mean CK-MB; the representation is the same as Figure 1.

CK and CK-MB isoenzyme during and after Exercise Testing (Salvadori et al)
first recovery could be due to myocardial distress with leakage of the enzyme from the cardiac musculature, which is similar to what already has been described in normal subjects subjected to massive physical stress without previous training.20

In the clinical setting, the amount of CK-MB isoenzyme in blood is expressed as a percentage of total CK and it is generally accepted that myocardial necrosis is more than 4 percent with a temporary elevation of total CK.21

Just considering the mean values of total CK of our obese patients, it may be possible that they have myocardial damage at percentages of CK-MB lower than those of lean subjects.

Attainment of a given heart rate is generally used in exercise testing for detecting myocardial ischemia.19 Considering our data, in obese heart patients, myocardial distress or damage may be present at a heart rate beneath the theoretic maximal rate so that further physical stress could be more useless than dangerous.

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