the 60 workers solicited for the study, 30 volunteered to submit to two consecutive blood drawings. The first sampling was at the beginning of the first morning shift after the week off. This was assumed to give baseline CK values. No instructions were given to the subjects concerning their physical activity during these days off. A control blood drawing was scheduled four working days later, at the end of the shift. The workers’ lean body mass was estimated from linear regression equations, with use of data on subcapsular and bicep skinfold thickness (4).

Total CK activity in the serum was measured at 37°C with the Boehringer "CK-NAC activated" test kit (no. 126349), for which normal CK values range from 24 to 195 U/L.

For five of these workers, the control CK values after four work days were >10 U/L lower than those measured before resuming work (range: −12 to −72 U/L). Evidently the recreational activities of these workers before the first CK determination induced higher CK concentrations than did the activity at the workplace itself. Therefore their CK values clearly could not be considered as baseline and were excluded from further analysis. Our results thus involved a total of 25 subjects. Table 1 lists their mean anthropometric characteristics, together with the CK values measured after the seven days off. The Spearman rank correlation coefficient was calculated, to determine the relationship between CK and lean body mass; the R coefficient was 0.36 (p <0.10). Moreover, we saw no relation between CK values and the lean body mass/body weight ratio.

The baseline CK values for these subjects were within the normal reference interval. This seems to correspond to the fact that none of them was involved in sports activities at a competitive level. We may thus consider these workers as "non-athletic" subjects. In this sample of a normal population, we saw no relation between baseline CK and lean body mass. These negative results contrast with those previously reported (9). The data suggest that the CK activity we measured before work was resumed was not actually baseline. The CK measured in these workers most likely involved two components: a basic component related to the subject’s muscle mass and an exercise component related to the type and intensity of recreational activity during the two days before the blood drawing. We conclude that a CK/lean-body-mass relationship can hardly be observed among non-athletes in normal life conditions when no limitation of the subjects’ physical activities can be enforced.

References

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Interference with PO2 Measurement in a Leukemia Patient

To the Editor:

We report a patient in whom an exceedingly low PO2 was recorded due to interference from leukocytes.

A 61-year-old male construction worker was admitted with a history of dyspnea and headache of 10 days duration. He had no history of chest pain or orthopnea. He was pale, jaundiced, and had an enlarged liver.

Hematological findings at the time of admission were: Hb 2.7 g/L, hematocrit 0.08, leukocytes, 186 × 10^6/L (blast cells 12%, myelocytes 2%, myelomonocytes 2%, and monocytes and promyelocytes 56%). Sternal marrow aspiration confirmed the diagnosis of myelomonocytic leukemia.

For an arterial blood sample, analyzed in a blood-gas analyzer (model IL1302, Instrumentation Laboratory), the pH was 6.61 and the PO2 was 36 mm Hg. A PO2 measurement could not be obtained because the PO2 value continued to decrease and even showed negative values. The calibration of the instrument immediately before analysis of the sample was satisfactory. The PO2 values for other patients, measured before and after this patient's sample, were compatible with their clinical conditions.

To identify the problem, we centrifuged the blood sample in a small capped tube and analyzed the plasma in the blood-gas analyzer; the PO2 was 60 mm Hg. When the cells were mixed with the plasma, the PO2 again was recorded as negative. A repeat arterial puncture was done later that night, but a positive blood PO2 value was not obtained. The blood-gas analyzer, checked thoroughly, was found to be satisfactory. The patient died later that night before any further investigation could be carried out. We suggest that this phenomenon of apparently very low PO2 is due to the obstruction of O2 diffusion across the membrane of the oxygen electrode by the extraordinarily large number of leukocytes present. We would be interested to hear of any similar experience.

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Misinterpretation of Study of Creatine Kinase BB

To the Editor:

Drs. Massey and Goe misinterpret our study in their discussion of serum creatine kinase BB isoenzyme activity (CK-BB) after cardiac arrest. We did not suggest as they state "that if CK-BB was detected within 6 h after the arrest the prognosis for complete recovery was poor" (1). Similar to their own results, we found that serum CK-BB was present in most patients (43 of 50, or 86%) resuscitated from cardiac arrest, regardless of neurologic outcome, when the sample was obtained within 6 h of the arrest (2). Rather, the persistence or reappearance of serum CK-BB more than 6 h after the arrest, in our series, was associated with poor neurologic recovery. Such was the finding in all patients without neurologic recovery, in 45% of those with incomplete neurologic recovery, but in only 4% of those with complete neurologic recovery.

Because of the many sources of CK-BB outside the brain that could cause