hemim, Mannheim, Germany) and the concentration of C-reactive protein (CRP; Behring, Marburg, Germany).

The relative increases in blood of total CK activities, CK-MB/CK ratios, and CRP and cTnT concentrations above the upper discriminator value are shown in Fig. 1 in an individual 25-year-old male patient with transient ST-segment elevation of >0.1 mV. Histological analysis of his myocardial biopsies revealed interstitial edema, perivascular vacuolization of sarcomplasmic reticulum, mild myocytolysis (by HE staining), and mononuclear T lymphocytes (by immunohistochemistry). In the total study group the endomyocardial biopsies of six patients revealed mononuclear infiltrates, with or without myocytolysis, classified as active or borderline myocarditis, respectively. Serum concentrations of cTnT were significantly increased, 2.8- to 60-fold, above the discriminatory value of 0.1 μg/L in all patients (N = 7) during the 2 to 8 days after onset of chest pain. Total CK activities were above normal (80 U/L, assayed at 25°C) in five patients during days 1 and 2. The CK-MB/CK ratio was >6% in only three of the seven patients.

In comparison with CK activities, serum cTnT provides a better sensitivity for detection of microinvasions in myocarditis because of a proportionally higher and longer-lasting increase in the blood. The data indicate that the cTnT assay may be a valuable tool to detect active or borderline myocarditis. Because all positive myocardial biopsies (six of seven) showed mononuclear cell infiltrates, future studies would be desirable to determine whether the patients with myocarditis and increased cTnT make up a subpopulation of patients with myocarditis and cellular infiltration.

Hypocatalasemia in Hospital Patients

To the Editor:

Hydrogen peroxide is involved in physiological phenomena and in the pathogenesis of various diseases [1-3]. Erythrocytes contain high concentrations of catalase (EC 1.11.1.6), which may regulate hydrogen peroxide either generated in erythrocytes or coming from other tissues [4, 5]. Erythrocytes are responsible for >98% of blood catalase. Despite the important function of catalase, studies of blood catalase activity in various diseases are few. Our aim in this paper was to measure blood catalase in a large group of patients with diagnosed diseases.

We measured blood catalase activity of all hospitalized patients (n = 28252) during a 3-year program. We classified the patients according to the final diagnoses at the time of discharge from our 350-bed hospital with internal medicine, surgery, neurological, and psychiatric departments. The procedures were in accordance with the ethical standards of our hospital's ethics committee. The blood catalase activity was measured with a spectrophotometric assay [6] adapted for blood samples [7]. The assay measures erythrocyte and plasma catalase combined, with the plasma component accounting for <1% of the total activity. The reference mean and range (±2SD) for blood catalase activity in presumably healthy subjects (n = 1753) were 113.3 MU/L and 80.3-146.3 MU/L, respectively. We used these values for comparison of catalase activities in patients with different diseases.

Hypocatalasemia (<80.3 MU/L) was present in 0.86% of the patients with different types of anemias, 0.29% of those with atherosclerosis, 0.27% with different types of tumors, and 0.09% with schizophrenia when all the patients were listed.

The 2884 anemic patients yielded a significantly lower blood catalase activity (83.7 ± 16.4 MU/L) than the reference value mean, with a correlation (r = 0.6954, intercept = 18.16, slope = 0.6823) between blood hemoglobin concentration and blood catalase activity. Hypocatalasemia, detected in 8.4% (iron deficiency: 78, hemorrhagic: 70, aplastic: 10, megaloblastic: 14, due to leukemia: 16, and chronic diseases: 55) of the anemic patients, could be explained by the decreased number of erythrocytes in these patients.

In 130 patients with different types of tumors (hematologic: 30, gastrointestinal: 29, lung: 17, urogenital: 14, digestive: 14, breast: 13, and miscellaneous: 13) and different IUCN classifications (mean values of T: 2.6, N: 2.3, and M: 2.1), the blood catalase activity (80.8 ± 21.5 MU/L) was significantly (P <0.001) below the reference mean, and 49.2% of these patients were hypocatalasemic. The blood hemoglobin concentration and blood catalase activity of these patients (Fig. 1) showed a correlation (r = 0.690, intercept = 17.03, slope = 0.573) different from that of anemic patients (slope = 0.6823). Therefore, the decreased blood catalase activity in these types of tumors may be attributed to the decreased production of erythrocytes, characterized by the mild anemia (blood hemoglobin in tumors 112.5 ± 18.9 g/L, P <0.001 vs 133.4 ± 11.6 g/L for healthy subjects), as well as to the decreased synthesis of erythrocyte catalase, verified by the lower values for slopes (0.573 vs 0.6823). These findings are in accordance with the results of Saito et al. [8] but different from those of Bewick et al. [9].

References


There are no published data on blood catalase activity in atherosclerosis. For these patients we found a significant ($P < 0.001$) decrease (104.8 ± 18.2 MU/L, $n = 1670$) in blood catalase activity with no change in blood hemoglobin concentration (135 ± 13 g/L). Hypocatalasemia affected 4.9% of these patients and showed a nonsignificant change with the seriousness of disease. Catalase activity was 102.6 ± 17.5 MU/L ($n = 660$) in the atherosclerosis affected coronary, cerebral, and other arteries and was higher in patients diagnosed with either cerebrovascular (105.1 ± 16.8 MU/L, $n = 509$) or coronary artery disease (107.3 ± 20.4 MU/L, $n = 501$).

In patients with schizophrenia, blood catalase activity was significantly ($P < 0.001$) decreased (105.6 ± 20.4 MU/L, $n = 275$) but the blood hemoglobin concentration was normal (136.4 ± 15.4 g/L), another new finding. Hypocatalasemia affected 9.2% of these patients and did not change when different types (paranoid, reactive, residual, simple) of this disease were examined.

Similarly to the Hungarian aca- talasemic patients [7], all of our hypocatalasemic patients did not yield any peculiar symptom that might be associated to their decreased blood catalase activity. Because of the high pathological frequencies involved, the contribution of hypocatalasemia to the signs and symptoms of tumors, atherosclerosis, and schizophrenia may not be excluded.

These findings do not suggest a diagnostic role for blood catalase determination. Rather, this test might be useful for selecting patients at risk and preparing them against situations associated with toxic amounts of hydrogen peroxide.

**References**


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**More on Magnum Automated T-Uptake Assay**

To the Editor:

As lead author of the recent report that evaluated five automated T-uptake assays [1], I would like to add several corrections and clarifications.

We reported that the Magnum Opus T-uptake assay (Behring Diagnostics, Westwood, MA) results were reported as $<15\%$ (the lower limit of the instrument’s dynamic range) in many of the patients with high concentrations of thyroid-binding globulin (TBG). Subsequently, Shai Inbar of Behring Diagnostics assured me that newer versions of the assay software (available by the second quarter of 1996) will have a wider dynamic range. He extrapolated each of the appropriate calibration curves from our data and calculated the T-uptake value for all the patients with increased TBG concentrations. The free thyroxine indices (FTI) were similar to those from the IMx method (Abbott Diagnostics, Abbott Park, IL), which we favored: 24 of 74 euthyroid individuals with increased TBG had decreased FTI values on the Magnum (compared with 15 of 74 with the IMx), and none had increased FTI values. No discrepant results were obtained with either assay in hyperthyroid or hypothyroid patients with increased TBG. The one increased TBG point for the Magnum in our Fig. 2b is an error, the result of a transcription error from the original assay. Finally, as stated in the Results section of the report, FTI was determined by multiplying total thyroxine by the thyroid hormone-binding ratio (THBR). This was true, however, only for those assays producing T-uptake results that, like the original T$_4$-uptake methods, are inversely proportional to the TBG concentration. For assays producing T-uptake results that are directly proportional to the TBG concentration (such as the IMx), the thyroxine should be divided by the THBR.

**Reference**


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