apo-AST with its coenzyme (5), while we (2) preincubated serum alone with 809 mmol of pyridoxal 5-phosphate per liter of 9 mmol/L phosphate buffer. Because we (2) used no asparagine in the preincubation mixture, and used six-fold more pyridoxal 5-phosphate and 10-fold less phosphate buffer, I would expect less inhibition of the recombination of apo-AST with its coenzyme in our assay system than anticipated by Hafkenscheid and Dijt.

The response of Drs. Hafkenscheid and Dijt regarding the ALT activity, similarly to that concerning the AST activity, does not deal with the issues raised in my letter and compares results previously reported by me (3) with those obtained with different analytical systems.

References
1–3. (Same as refs. 1–3 in Lustig’s Letter, above).

Viliam Lustig

Glutamyltransferase Activity during Pregnancy

To the Editor:

Brocklehurst and Wilde recently published an interesting article in Clinical Biochemistry (26/5, 588–591, 1980) on alkaline phosphatase, glutamyltransferase (GTP), and 5'-nucleotidase activities in amniotic fluid during pregnancy.

We found very interesting the clinical value of the determination of alkaline phosphatase in the evaluation of fetal maturity, but regarding the measure of γ-GTP activity in amniotic fluid throughout gestation, the authors reported that they had no knowledge of any previous work.

We have published two earlier papers on this subject (1, 2). The results of Brocklehurst and Wilde agree with ours concerning the activity of γ-GTP during gestation. Moreover, the great difference between the enzyme activities in amniotic fluid at the beginning and at the end of the gestation is ascribable to the presence of two different γ-GTP iso-enzyme forms (2).

References

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Evaluation of Serum Monoamine Oxidase in Patients with Connective Tissue Diseases

To the Editor:

Biochemical studies on collagen synthesis (1, 2) suggest that a monoamine oxidase [EC 1.4.3.4; amine oxidase (flavin-containing)] catalyzes the maturatio of connective tissue fibers, specifically the cross linking of both collagen and elastin. An increased serum monoamine oxidase activity has been reported in hyperthyroidism (3), in chronic liver diseases (4), congestive heart failure (5), and diabetes mellitus (6). The highest activities so far have been found in liver cirrhosis and reportedly depend on fibrotic processes (7).

Because activities of this enzyme in serum have been related more specifically with fibroblast synthesis, we evaluated it in 20 patients with connective tissue diseases: seven with progressive systemic sclerosis (PSS), five with systemic lupus erythematosus (SLE), and eight with rheumatoid arthritis (RA). In all the cases, the diagnosis was also supported by radiological (RA) and histological (SLE and PSS) features. We used the “MAO Test” (Wako-Italfarmaco), a colorimetric method based on measurement of benzaldehyde formed by an enzyme reaction with a benzylamine derivative as substrate (8). Reference intervals obtained from data on 209 clinically healthy subjects were 4–39 U/L (mean ±2 SD). Different values were found in the examined connective tissue diseases, as shown in Figure 1.

The PSS patients with cutaneous, esophageal, and enteric involvement generally showed the highest values; lower values were present in patients with only cutaneous lesions. Of five SLE patients (all with renal damage) only three had values of about 50 U/L. Of the eight patients with RA, only one showed a slight increase in monoamine oxidase activity.

We saw no correlation between enzyme activity and immunological and (or) laboratory features of inflammatory diseases such as increased α2- and γ-globulins and increased erythrocyte sedimentation rate. Increased activity is probably related to the fibrotic process, which is markedly greater in PSS than in RA or SLE, according to Leroy (9), who observed in vitro a primary abnormality of connective tissue (a de-

References

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Fig. 1. Distribution of serum monoamine oxidase activities in 20 patients with connective tissue diseases and in 209 healthy subjects. The three horizontal lines indicate the mean ±2 SD.
fect in the regulation of collagen synthesis at the fibroblast level). Obviously, this determination alone is not sufficient for a differential diagnosis, because monoamine oxidase also is present in non-connective tissues; serial determinations, however, could be useful in evaluating the activity of the disease.

We thank Dr. Fabio Ayala, M.D., Dept. of Dermatology, 2nd School of Medicine, Naples, for his kind collaboration.

References

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Serum Nickel in Myocardial Infarction

To the Editor:

Following the recent Case Report (1), involving conflicting electrocardiographic and enzyme studies, you published a Letter (2) pointing out the possible value of serum zinc measurements in diagnosing myocardial infarction.

In this laboratory, trace-element determinations have proved valuable in resolving diagnostic difficulties encountered in cardiac enzyme profiling. While the change in serum zinc concentrations are reflected in some of my data, I find that serum nickel measurements provide a more definitive diagnostic aid.

Sunderman and his co-workers (3, 4) have investigated serum nickel after myocardial infarction, and they conclude that it is frequently increased. An exchange of serums between Sunderman’s laboratory (3) and this department revealed good correlation for the increases in nickel after infarction. Differing absolute values were obtained, owing to methodological factors which have since been resolved. The normal range determined in this laboratory is up to 5.0 μg of Ni per litre of serum, which is close to Sunderman’s range (1.0 – 4.2 μg/L).

Serum nickel can be measured by atomic absorption (5), although an emission method is used here.

Table 1 summarizes our results, which show the increase in serum nickel after myocardial infarction, together with control group values.

We determine serum nickel in all cases of possible myocardial infarction where diagnostic difficulties are present and find the results consistently helpful.

References

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Table 1. Serum Nickel Concentration in a Control Group and Patients with Myocardial Infarction

<table>
<thead>
<tr>
<th>No. subjects</th>
<th>Serum Ni (μg/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
</tr>
<tr>
<td>Healthy controls</td>
<td>123</td>
</tr>
<tr>
<td>Hepatic disease</td>
<td>55</td>
</tr>
<tr>
<td>Hospital control</td>
<td>68</td>
</tr>
<tr>
<td>Acute myocardial infarction, h post-episode</td>
<td>54</td>
</tr>
<tr>
<td>0–6</td>
<td>5</td>
</tr>
<tr>
<td>6–12</td>
<td>8</td>
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<td>12–24</td>
<td>20</td>
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<tr>
<td>24–36</td>
<td>32</td>
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<td>36–48</td>
<td>23</td>
</tr>
<tr>
<td>48–72</td>
<td>28</td>
</tr>
<tr>
<td>72–96</td>
<td>17</td>
</tr>
<tr>
<td>96–120</td>
<td>6</td>
</tr>
<tr>
<td>120–216</td>
<td>27</td>
</tr>
</tbody>
</table>

Myocardial ischemia without infarction | 12 | 3.2 | 1.9–4.2 |

Reference Intervals for Ferritin: Age Dependence

To the Editor:

During the evaluation of a new assay for ferritin (Ventrex Laboratories, Inc., ME 04103) it became apparent that ferritin concentrations were increased in normal menopausal women and, to a lesser extent, in men over 45 years of age. Although Valberg et al. (1) investigated to some extent the effects of age, the grouping was such that it did not differentiate menopausal from premenopausal women, or men over or less than 45 years of age. In addition, it appears that the authors used parametric statistics of evaluate their data, whereas we found it necessary to apply the nonparametric statistical method of Herrera (2).

Our findings are summarized in Table 1. In most instances a complete blood