precision of the measurement was considerably improved by 
only a short oral explanation of possible trivial errors 
(Figure 1B). In this case about 95% of the operators mea-
sured C_{Hb} with a relative error (CV) of <3.5%, which 
comparres favorably with results reported elsewhere (4).

We conclude that the Hemocue is a reliable, rapid, and 
convenient instrument system for measurement of C_{Hb} in 
whole blood, particularly suitable for urgent determinations 
and for use in general practice. (Marketing and technical 
service in the United States is available exclusively through 
Medical Equipment Designs, Inc., 23461 Ridge Route Drive, 
Suite F, Laguna Hills, CA 92653.)

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determination of clinically significant hemoglobin derivatives by 

Measurement of Progesterone Receptors in Breast-
Tumor Cytosols by Immunoenzymatic Assay: 
Preliminary Results, T. Metatyé, J. Y. Bounaud, and F. 
Begon (Lab. de Biophysique Cellulaire, Service de Méd. 
Nucléaire, Hôpital Jean Bernard, BP 577, 86021 Poitiers 
Cedex, France)

A new enzyme immunoassay (Abbott's PgR-EIA Monoclo-
nal) for determination of progesterone receptors in cytosols 
from breast-tumor tissues was recently proposed by Abbott 
Laboratories, Rungis, France. This assay, based on a direct 
antigenic binding, includes two monoclonal antibodies to 
the receptors: one immobilized on beads, the other labeled 
with horseradish peroxidase, the enzymatic reagent. This 
assay was tested in our laboratory and results were 
compared with those by our multipoint dextran-coated charcoal 
method (DCC), in which titrated promegeston ([\(^{13}H\)-R5020) 
is used as ligand.

Cytosols (n = 31) prepared with Tris buffer containing 
KCl (0.4 mol/L) were consecutively assayed by the two 
tests. Ten had receptor concentrations below the limit of 
detection (3 fmol per milligram of cytosol protein) by DCC, 
but ranged between 1 and 18 fmol/mg by PgR-EIA (mean 8, 
SD 6 fmol/mg). Values for 21 ranged from 3 to 51 fmol per 

milligram of cytosol protein (mean 21, SD 15 fmol/mg) by 
DCC but gave concentrations ranging between 2 and 261 
fmol/mg by PgR-EIA (mean 118, SD = 96). Linear regression 
analysis for progesterone receptor concentrations as 
measured with the PgR-EIA (\(y\)) and DCC (\(x\)) assays gave: \(y = 5.1x + 11.2\) (\(r = 0.85, n = 31, P < 0.001\)). Although results 
by the two methods correlated well, the concentrations 
measured by PgR-EIA were fivefold those by our DCC 
method. This can probably be attributed in part to the 
methodological difference between the two methods: the 
DCC assay measures active steroid binding sites; the PgR-
EIA assay measures an antigenic activity.

We think that the PgR-EIA assay represents a good 
alternative to the multipoint DCC method: the assay is easy 
to perform in routine laboratory practice and gives results 
that correlate well with the DCC method. Nevertheless, 
more investigations should be performed to examine the 
hormonal dependency of tumors on the concentrations of 
progesterone receptors obtained by PgR-EIA.

Mannitol Interference in an Automated Serum 
Phosphate Assay, A. Bradley Eisenbrey, Ranjit Mathew, 
and Frederick L. Kiechle (Dept. of Clin. Pathol., William 
Beaumont Hospital, 3601 West Thirteen Mile Road, 
Royal Oak, MI 48072)

Mannitol is used clinically to reduce intracranial pressure 
temporarily in patients with brain edema, and as an osmotic 
diuretic agent. Mannitol interference with assay of inorgan-
ic phosphate in serum has been reported, as has inhibition 
of the color development of phosphomolybdate in the DuPont 
"aco" method—but not in some other instruments (1–4).

We received a serum sample from a 72-year-old woman 
who developed a subdural hematoma after a fall. Therapy 
included intravenous mannitol. Serum phosphate, deter-
mined in the American Dade "Paramax" after initiation of 
therapy, was 0.16 mmol/L (normal 0.80—1.60 mmol/L). Val-
ues for other serum analytes—including calcium, total 
protein, and albumin—were normal, and no explanation for 
hypophosphatemia was found on reviewing the chart. There 
was insufficient sample for repetition by an alternative 
method. We suspected mannitol interference in phosphate 
assay in the "Paramax" and did the following experiment, 
adding various amounts of aqueous mannitol to five sera 
such that final volumes were constant, then assaying (ND: 
not done).

<table>
<thead>
<tr>
<th>Mannitol, mmol/L (final concn)</th>
<th>Measured phosphate concn, mmol/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>0.74 1.52 1.42 1.00 1.00</td>
</tr>
<tr>
<td>B</td>
<td>0.68 1.52 1.45 1.00 1.00</td>
</tr>
<tr>
<td>C</td>
<td>0.85 1.52 1.39 0.99 0.94</td>
</tr>
<tr>
<td>D</td>
<td>0.61 1.42 1.29 0.87 0.87</td>
</tr>
<tr>
<td>E</td>
<td>0.55 1.42 1.29 0.84 0.84</td>
</tr>
<tr>
<td>137</td>
<td>ND ND ND 0.84 0.81</td>
</tr>
</tbody>
</table>

Samples assayed in the American Monitor "Parallel" 
showed no effect of mannitol on the measured phosphate. All 
commonly used automated methodologies are based on 
formation of molybdenum blue from the phosphomolybdate 
complex. Differences in the susceptibility to mannitol inter-
ference may be explained by the different reducing agents 
used in the procedures, which may make the Paramax and 
aco (3, 4) more sensitive to mannitol interference.

No therapeutic intervention to correct the pseudo-
hyponatremia was undertaken in this patient. Patients 
receiving mannitol may receive unnecessary phosphate, 
risking hypocalcemia and stone formation, if treatment is 
guided by serum phosphate concentrations reported from 
the aco or Paramax.

References
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Significance of Increased Serum Lactate Dehydrogenase Isoenzyme 5 in Acute Infero-Posterior Myocardial Infarction, Zvi Rotenberg, Izhak Weinberger, Ehud Davidson, Jacob Fuchs, Oded Sperling, and Jacob Agmon (Israel and Ione Massada Center for Heart Diseases, Dept. of Med. "A"; and 1 the Clin. Biochem. Lab., Beilinson Medical Center, Petah Tikva 49100; and the Tel Aviv Univ. Sackler School of Med., Tel Aviv, Israel. Address correspondence to J.A. at the BMC)

In 30–60% of patients with acute infero-posterior myocardial infarction (AIPMI), right ventricular (RV) infarction may be diagnosed when overt signs of right ventricular dysfunction (RVD), such as jugular venous engorgement and liver congestion, are present. An enzyme abnormality commonly seen in congestive heart failure is increased lactate dehydrogenase (LD) and disproportionally high isoenzyme LD-5, probably as a result of passive liver congestion and cellular hypoxia (J–3). We wanted to determine the LD-5 proportions in patients with AIPMI with and without RVD, and to see if RVD and increased serum LD-5 values are correlated.

We studied 30 consecutive patients admitted to our coronary-care unit and medical department with a first transmural AIPMI. On admission each patient was clinically assessed for signs of RVD—jugular venous engorgement, Kussmaul's sign, and liver congestion. Hemodynamic status was classified according to the criteria of Killip and Kimball (4). According to echocardiographic and isotope angiographic studies performed in each patient, two groups could be defined: (a) 40 patients with AIPMI and RVD (right ventricular wall motion abnormalities on echocardiography and low right ventricular ejection fraction); and (b) 50 patients with AIPMI and normal right ventricular function.

Total serum LD activity and LD isoenzymes were determined in each patient 24, 48, and 72 h after admission for chest pain.

We compared clinical and enzymatic variables in these two groups. Differences between means, assessed by Student's t-test, were considered significant at P <0.05 (Table 1).

Mean values for creatine kinase and LD values were not significantly different between the two groups. Peak LD-1/LD and LD-4/LD ratios were not significantly different between the two groups of patients, but the peak LD-5/LD ratio 48 h after admission was significantly higher, 0.11 ±0.03, in the RVD group of patients than in the non-RVD group, 0.02 ±0.03 (P <0.01). In 28 of 40 patients with RVD the peak LD-5/LD ratio was >0.10; such a high ratio was not found in any of the non-RVD patients.

A possible explanation for the increased LD-5/LD ratio in the RVD patients may be a subclinical congestive heart failure associated with RVD, causing liver congestion without overt clinical signs of hepatomegaly (5–6). Chapelle et al. (7) described an increased LD-5/LD ratio in about one quarter of 385 patients with acute MI, most of them with AIPMI, but there was no correlation between RVD and an increased LD-5/LD ratio.

Our results indicate that in patients with AIPMI and RVD, an increased LD-5/LD ratio may be found even in the absence of overt clinical signs of RVD, and may thus be an indicator for right ventricular infarction in patients with AIPMI.

References

Table 1. Clinical and Enzymatic Variables in the Patients

<table>
<thead>
<tr>
<th></th>
<th>RVD</th>
<th>Non-RVD</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>40</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>Age, y (mean ± SD)</td>
<td>60 ± 8.1</td>
<td>62 ± 9.0</td>
<td>NS</td>
</tr>
<tr>
<td>Venous engorgement (no. pts.)</td>
<td>20</td>
<td>12</td>
<td>NS</td>
</tr>
<tr>
<td>Kussmaul's sign (no. pts.)</td>
<td>18</td>
<td>2</td>
<td>P&lt;0.05</td>
</tr>
<tr>
<td>Liver congestion (no. pts.)</td>
<td>8</td>
<td>2</td>
<td>P&lt;0.01</td>
</tr>
<tr>
<td>Killip, mean ± SD</td>
<td>1.5 ± 0.77</td>
<td>1.3 ± 0.57</td>
<td>NS</td>
</tr>
<tr>
<td>CK, peak value (U/L), mean ± SD</td>
<td>950 ± 550</td>
<td>1150 ± 600</td>
<td>NS</td>
</tr>
<tr>
<td>N &lt;200 U/L</td>
<td>720 ± 270</td>
<td>830 ± 310</td>
<td>NS</td>
</tr>
<tr>
<td>LD, peak value (U/L) mean ± SD</td>
<td>39 ± 0.31</td>
<td>42 ± 0.07</td>
<td>NS</td>
</tr>
<tr>
<td>N 0.1–0.31</td>
<td>0.37 ± 0.03</td>
<td>0.40 ± 0.04</td>
<td>NS</td>
</tr>
<tr>
<td>LD-2/LD peak, mean ± SD</td>
<td>0.10 ± 0.02</td>
<td>0.12 ± 0.02</td>
<td>NS</td>
</tr>
<tr>
<td>N 0.0–0.06</td>
<td>0.03 ± 0.01</td>
<td>0.04 ± 0.02</td>
<td>NS</td>
</tr>
<tr>
<td>LD-3/LD peak, mean ± SD</td>
<td>0.11 ± 0.01</td>
<td>0.02 ± 0.03</td>
<td>P&lt;0.01</td>
</tr>
<tr>
<td>LD-4/LD peak, mean ± SD</td>
<td>none</td>
<td>P&lt;0.01</td>
<td></td>
</tr>
<tr>
<td>LD-5/LD &gt;0.10, no. pts.</td>
<td>28</td>
<td>P&lt;0.01</td>
<td></td>
</tr>
</tbody>
</table>

N: normal reference interval.