with technetium-99 pyrophosphate (Tc-PYP-ECT) and thallium-201 (TI-ECT) were also undertaken, as well as repeated ECG recordings.

Our reference interval for CRP was 0–10 mg/L and for CK and CK-MB 0–250 U/L and 0–25 U/L, respectively. All patients had a diagnostically useful increase in CK and CK-MB in serum, with mean peak values of 2532 (SD 1450) U/L and 365 (SD 185) U/L, respectively. All also had positive findings diagnostic for MI in their Tc-PYP-ECT examinations.

Seven patients had markedly increased serum CRP concentrations (mean of peak values 165 (SD 51) mg/L), which began to increase 24 (SD 9) h after onset of pain and peaked after 83 (SD 30) h. In three patients the previously normal values for serum CRP did not increase.

Thus, most of our MI patients, who all fulfilled the wto criteria for MI, had a marked increase in serum CRP, but some did not. The latter patients differed from the CRP-positive patients in not having a diagnostic finding for MI in the TI-ECT examination and not developing QRS changes in their ECGs. They also, on the average, had smaller infarcts than did the CRP-positive patients. However, the size of the infarct alone did not seem adequately to explain the lack of CRP response in our CRP-negative patients. Indeed, the patient with the smallest increases in CK showed an increase in CRP up to a value of 128 mg/L.

There was a relatively long lag-phase, as compared with (e.g.) bacterial infections, before the CRP increased in the CRP-positive patients. This may indicate that the CRP response was not due directly to ischemic cell death but rather to some later event in infarct development that did not take place in the CRP-negative patients. Perhaps, infarct maturation in the intermediate subendocardial layer differs from that of the deeper layers of the myocardium and leads to qualitatively different CRP responses in subendocardial and transmural infarcts. The concept of a subendocardial infarction in our CRP-negative patients would agree with their negative TI-ECT results and the lack of QRS changes in their ECGs.

We conclude that serum CRP may fail to increase in acute MI and that, if so, this indicates a mild and probably subendocardial infarction.


This study was undertaken to ascertain whether commercial B12 radioassay kits can measure low concentrations of cobalamin in the presence of a large excess of cobinamide. Three serum pools were assayed: one normal (450 pg/mL) and two with subnormal B12 concentrations (<160 pg/mL), each divided into three subpools. Subpool A contained 396 pg of added cyanocobalamin per milliliter, for recovery assessment. Subpool B contained 9.897 ng of added cobinamide dicyanide per milliliter for analog cross-reactivity studies. Subpool C was baseline. We tested 14 radioassay B12 kits from seven manufacturers. In 11, purified intrinsic factor is used as binder, and in three R-proteins–IF mixtures are used, two blocked and one non-blocked. Serum protein is denatured by heat (in eight kits) or by alkali (in six kits). In nine kits charcoal is used for bound–free separation and in five solid-phase separation is used. The mean B12 values found for 12 of the kits were:

<table>
<thead>
<tr>
<th>Pool</th>
<th>Subpool C</th>
<th>Subpool B</th>
<th>Recovery, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>450 (CV 10.6%)</td>
<td>433 (CV 10.5%)</td>
<td>94.4 (CV 20.0%)</td>
</tr>
<tr>
<td>2</td>
<td>136 (CV 19.1%)</td>
<td>140 (CV 19.8%)</td>
<td>101 (CV 12.0%)</td>
</tr>
<tr>
<td>3</td>
<td>150 (CV 17.2%)</td>
<td>156 (CV 18.4%)</td>
<td>104 (CV 9.2%)</td>
</tr>
</tbody>
</table>

One kit's results were discarded because the zero and 50 pg/mL standard counts/minute were indistinguishable for two lots tested.

Although the CVs indicate significant between-kit differences for B12 found, they can, as a group, distinguish between cobalamin and cobinamide at low and normal B12 concentrations and quantitatively account for cobalamin.

Precipitation of Multiple Myeloma Serum in the Phosphorus Channel of the Beckman Astra, Philip L. Tipper and Don S. Miyada (Department of Pathology, University of California Irvine Medical Center, Orange, CA 92668)

We use the Beckman Astra phosphorus module to measure phosphorus in serum. The Beckman method uses ammonium molybdate in 1 mol/L sulfuric acid (1).

Reported values of 0 mg/L for serum from a 45-year-old woman led us to investigate. The diagnosis was multiple myeloma. Values for total protein in the patient's serum ranged from 135 to 150 g/L during this admission. Values for phosphorus obtained with the Du Pont acc were about 30 mg/L.

After the analysis of her serum, the Astra phosphorus module cup contained a white, gelatinous precipitate that adhered to the wall of the cup and to the magnetic stirrer. A similar precipitate formed when we added a drop of the patient's serum to 1 mL of the Astra phosphorus reagent in a test tube.

We have observed this phenomenon before when sera from myeloma patients were subjected to highly acidic conditions, e.g., in the o-toluidine method for glucose used with the Technicon SMA 6/60.

In addition to the spurious effect on the myeloma patient's phosphorus results, this phenomenon may also affect the analysis of other patients' samples because the precipitate remains in the analysis cup.

Users of the Beckman Astra phosphorus module should use care when analyzing the serum of patients with high total protein content, and should examine the equipment for precipitates after analyzing such sera. Users of other instruments that involve highly acidic reagents may wish to take similar precautions.

Reference