jointly than individually. There can be a physiological basis for this. If the raised level of myocardial enzymes is the direct consequence of myocardial necrosis (Sobel et al, 1972) and the raised level of FDP indicates a fibrinolytic response to a previous thromboembolic episode (Merskey and Johnson, 1971) then the patient should be suffering from an extensive myocardial lesion and its consequential thromboembolic complications. Interestingly, none of our three patients who had a raised FDP level with low enzyme levels died. Therefore perhaps the opinion that a high FDP level is a sign of a poor prognosis in acute myocardial infarction should be revised unless it is also associated with raised myocardial enzymes.

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References

Rapid estimation of paracetamol in plasma

Many methods for the determination of paracetamol in serum and plasma have been described in recent years, and of these the method of Routh et al (1968) has gained widespread acceptance. The method uses the differential absorbance at 266 nm of an acidified and alkalized extract in order to minimize interference by salicylate. This choice of analytical approach means that the method is relatively insensitive (< 100 μmol/l) and that both barbiturates and phenylbutazone interfere. We encountered five cases of mixed overdose involving paracetamol and barbiturate in 1975.

Dordoni et al (1973) described a rapid method for the determination of paracetamol to which they ascribed a lack of specificity and sensitivity. Contrary to these authors we have found that, apart from being extremely rapid, this method is more sensitive and less susceptible to interference by other commonly abused drugs than is the method of Routh (1968), but the presence of high concentrations of phenylbutazone may give misleading results (table).

The method used in our laboratory is as follows:
To 1 ml serum or plasma add 1 g NaCl (approx) and 10 ml diethyl ether (Analar). Mix gently for 30 seconds by inversion, and as it is very rare for emulsions to form the ether may be decanted directly into a quartz cuvette and the absorbance read at 250 nm against an ether blank. A serum-based standard should also be taken through the procedure.

The assay is linear to 1000 μmol/l. Precision of the method was excellent (CV = 1-8% at 750 μmol/l, N = 16). One possible objection to the method is that falsely high results might be obtained due to evaporation of the ether. This has been negligible in our experience, the absorbance of paracetamol in ether increasing by less than 0-5% per minute at 25°C and by 1% per minute at 37°C.

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References

<table>
<thead>
<tr>
<th>Drug</th>
<th>Concentration (μmol/l)</th>
<th>Interference (as μmol/l paracetamol)</th>
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<tbody>
<tr>
<td>Phenobarbitone</td>
<td>400</td>
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</tr>
<tr>
<td>Salicylate</td>
<td>10</td>
<td>25</td>
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<tr>
<td>Methaqualone</td>
<td>400</td>
<td>50</td>
</tr>
<tr>
<td>Phenylbutazone</td>
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<td>200</td>
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<tr>
<td>Glutethimide</td>
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</tr>
<tr>
<td>Carbromal</td>
<td>850</td>
<td>0</td>
</tr>
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</table>

Table: Effect of the presence of other drugs on the determination of paracetamol.