Letters to the Editor

Table 1  Comparison of chlamydia culture and enzyme immunoassay using rectal swabs

<table>
<thead>
<tr>
<th>Enzyme immunoassay</th>
<th>Culture</th>
<th></th>
<th>Contaminated</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Positive</td>
<td>Negative</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive</td>
<td>1</td>
<td>16</td>
<td>1</td>
<td>18</td>
</tr>
<tr>
<td>Equivocal</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Negative</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Total</td>
<td>1</td>
<td>19</td>
<td>3</td>
<td>23</td>
</tr>
</tbody>
</table>

Four of the five Staphylococcus aureus strains gave positive results, whereas the S epidermidis strain was negative. Positive results were also produced by a preparation of protein A. It seems unlikely, however, to be the explanation for false positives with faeces and rectal swabs, as when serial dilutions of S aureus were tested it was established that an inoculum of 10^3 organisms/ml was required to produce positive results. This is clearly far larger than the likely numbers of S aureus in either faeces or rectal swabs.

We have therefore been unable to identify the precise mechanism of the false positive results, but it would seem likely that it entails a non-specific binding to mouse immunoglobulin, as in our studies prior absorption of faeces with mouse serum gave a two fold reduction in reading in the enzyme immunoassays, compared with absorption in transport medium.

We have shown that this kit is quite unsuitable for examining rectal swabs, although in fairness the manufacturer’s instructions indicate that it is designed for examining genital specimens only. We consider, however, that this problem should be investigated further to ensure that it cannot produce occasional false positive results, with genital specimens. Even if false positive results were uncommon, they would assume importance if the kit were used to screen populations, such as antenatal clinics, which may have a low prevalence of true positive results, rather than the high incidence in the populations examined in reported studies.

Table 2  List of faecal organisms tested by enzyme immunoassays

| Acinetobacter calcoaceticus var anitratus |
| Bacteroides fragilis |
| Bacteroides melaninogenicus |
| Bacteroides ovatus |
| Escherichia coli |
| Fusobacterium fusiforme |
| Klebsiella aerogenes |
| Salmonella typhimurium |

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References
In a healthy subject HPV infection can induce a transient arrest of erythropoiesis, which does not lead to clinical symptoms because the normal erythrocyte life span exceeds that of any effect of HPV on marrow. In patients with haemolytic anaemias the haemoglobin concentration drops greatly with HPV infection, because the red blood cell life span is shortened. In our case the apparent HPV aplastic crisis was similar to those in previous reports, but erythroblastopenia associated with HPV in non-haemolytic anaemia has not been reported as far as we know. It seems that the reticulocytopenia induced by HPV infection can thus worsen a pre-existing anaemia. Our bone marrow findings also showed that HPV, besides its known erythroblastopenic effect, can cause a moderate and transitory decrease in the platelet count, secondary to a hypoplastic effect on the megakaryocytes. Our patient did not have any erythema arthralgia, or vascular purpura, all of which may be seen in HPV infection.*  

We suggest that when a patient with a haemolytic or any other type of anaemia, presents with a rapid and unexplained fall in peripheral blood haemoglobin concentration compared with steady state values, it seems logical to look for HPV infection.  

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References  

Evaluation of strategy for testing thyroid function applied to hypothyroidism  
In their interesting account of an evaluation of a strategy for thyroid function testing, Corns and Miller* illustrate the particular importance of relevant clinical information accompanying requests to the laboratory.  

Tests for thyroid function services have been changing rapidly over the past two decades, and in many hospitals the laboratory has had to take the initiative in the selection of appropriate tests for individual patients. Various strategies have been argued recently, with agreement that a cost effective laboratory service can be achieved if there is cooperation between clinician and pathologist.  

For 15 months we have been operating a thyroid function strategy based on ideas of Britton et al., but using free thyroxine (Amerlex-M Free T4 RIA kit) as the primary screening test and thyroid stimulating hormone as a discretionary test. The strategy requires a set of decision aiding reference ranges and levels. For detection of hypothyroidism, the relevant values were: euthyroid reference range 9.9–23.7 pmol/l (0.77–1.84 ng/100 ml); a therapeutic reference range for patients receiving thyroxine 12.9–30.8 pmol/l (1.0–2.39 ng/100 ml), and a further decision aiding range 12.3 pmol/l (0.9 ng/100 ml) to select thyroid stimulating hormone in patients presenting with features of hypothyroidism. Thyroid stimulating hormone was also assayed with free thyroxine as the primary investigation, whether or not requested in the following: paediatric cases; patients receiving lithium, amiodarone, or fenclofenac; pregnant patients; those with carcinoma of the thyroid; and goitre with antibodies (we called all these “the problem group.”)  

Continuous review of our results over 12 months showed that: (i) 22 cases of thyroid stimulating hormone were not tested because, in the absence of clinical details, the patient had not been allocated to the “problem group.” Subsequent testing showed that nine of these patients had raised thyroid stimulating hormone values. (ii) Fifty six patients with low free T4 values had normal thyroid stimulating hormone results, and all of these were acutely sick ill patients with no features of thyroid disease. (iii) Three patients with repeatedly normal serum free T4 concentrations had consistently raised thyroid stimulating hormone values. They all had clinical features of hypothyroidism. On subsequent testing clinically important titres of antithyroxine antibodies were detected in the sera (anti-T3 was also detected in one patient). Reassay of the hormone after precipitation of interfering immunoglobulin gave more accurate free T4 values (table).  

We agree that relevant clinical details with requests for thyroid function testing are necessary to the successful management of a laboratory testing strategy.  

Screening for thyroid disease in acutely ill patients with no suspicion of thyroid disorder seems to be wasteful of essential resources, and the results can be misleading. Accordingly, we requested house doctors not to screen patients during acute illness.  

It seems that interfering antithyroid antibodies may occur more commonly than originally suspected, and both laboratory staff and clinicians should be alert to this possibility when confronted with anomalous results.  

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References  

| Table | Thyroid function strategy |  |
|---|---|---|---|---|---|---|---|
| Case No | First free T4 (pmol/l) | Thyroid stimulating hormone (pmol/l) | Percentage counts precipitated as antibody | Supernatant free T4 (pmol/l) | Clinical details |
| 1 | 24.7 (1.92) | > 60 | | 3.6 (0.3) | “Puffy face” looking myxoedematous |
| 2 | 11 (0.85) | 15.5 | 11 | 8 (0.62) | Gaining weight, warm, and became thyroid |
| 3 | 10 (0.78) | 31.9 | 13 | 4 (0.35) | Deep voice, slowing up, myeloma |

Patients with circulating antithyroxine antibody (values in parentheses given in ng/100 ml).