Letters to the Editor

having a higher sensitivity and the same specificity (100%) as the CLO-test. Our results indicate that a positive CP-test before 20 minutes' incubation is strong evidence of C pylori infection and is an indication for treatment. Furthermore, if a urease test becomes positive after one hour of incubation then treatment should not be started immediately as other urease producing species may be responsible for the positive test, and confirmation for the presence of C pylori should be obtained either by histological examination or culture.

Table Results of tests for HIV antibody and antigen in laboratory serum control reagents found positive for anti-HIV in preliminary screening enzyme immunoassay

<table>
<thead>
<tr>
<th>HIV antibodies</th>
<th>Enzyme immunoassay</th>
<th>Confirmatory</th>
<th>Immunofluorescence assay</th>
<th>HIV antigen(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum control reagents*</td>
<td>Screening (ratio of optical density, cut off)</td>
<td>Anti-core P24</td>
<td>Anti-ENV GP41</td>
<td>(HIV-infected H9 cells as substrate)</td>
</tr>
<tr>
<td>1</td>
<td>5.99</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>2</td>
<td>5.02</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>3</td>
<td>3.19</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>4</td>
<td>2.22</td>
<td>+</td>
<td>+</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>1.81</td>
<td>+</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>6</td>
<td>1.73</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>7</td>
<td>1.71</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>8</td>
<td>1.54</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>9</td>
<td>1.37</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

+ positive result; 0, negative result.
*Serum lots 1, 2, 4 and 6 from manufacturer I; 3, 8 and 9 from manufacturer II; 5 and 7 from manufacturer III.

 manufacturers, which are devised for the calibration of biochemical tests for evidence of contamination with HIV. None of the CSF serum control samples contained antibodies to HIV but nine lots of controls purchased between September 1984 and December 1986 from three manufacturers were found to be reactive in a commercial (Abbott) enzyme immunoassay (EIA). Eight of these results were verified in the Abbott "confirmatory" EIA for anti-HIV. One of the reagents was positive, another weakly positive, and two gave equivocal reactions when tested by immunofluorescence in H9 cells infected with HIV. HIV antigen was not detected in any of the antibody-containing lots by the Abbott EIA for HIV antigen(s) (table). One of the manufacturers told us that reverse transcriptase could not be detected in cultures of H9 cells inoculated with their serum control lots. Nevertheless, as donors infected with HIV contributed to the pools from which the serum controls were prepared, these reagents should be regarded as contaminated with HIV and therefore potentially infectious for laboratory personnel.

Notwithstanding the lack of evidence of inadvertent infection of laboratory technicians with HIV and the possible inactivation of the virus during preparation of these controls, we take the view that reagents for laboratory use should be prepared from sera non-reactive for anti-HIV. We are assured by the manufacturers whose products we tested that they now follow this practice.

Within the past few weeks we have received a further consignment of eight lots of serum control samples, one of which was repeatedly reactive in the Abbott EIA for anti-HIV. This lot, supplied by a manufacturer whose products we had not tested previously, was of low reactivity and found to be negative in the Abbott confirmatory test.

We would endorse the recommendation of the Centers for Disease Control, Atlanta, that human sera used as controls or reagents should carry a caveat to the effect that freedom from contamination with HIV cannot be guaranteed. Additionally, it should be stated whether the product has been tested for the presence of anti-HIV or HIV antigen, and, if the test has been done, the result should be given.

Anti-human immunodeficiency virus (HIV) positive laboratory reagents

In June 1985 Jones et al drew attention to the potential infectivity for acquired immune deficiency syndrome (AIDS) of laboratory reagents used in the diagnosis of bleeding disorders; antibody to the human immuno-deficiency virus (HIV) was detected in eight of 15 laboratory reagents prepared from human plasma. We examined 30 lots of human serum and six control samples of cerebrospinal fluid (CSF), from eight manufacturers, which are devised for the calibration of biochemical tests for evidence of contamination with HIV. None of the CSF serum control samples contained antibodies to HIV but nine lots of controls purchased between September 1984 and December 1986 from three manufacturers were found to be reactive in a commercial (Abbott) enzyme immunoassay (EIA). Eight of these results were verified in the Abbott "confirmatory" EIA for anti-HIV. One of the reagents was positive, another weakly positive, and two gave equivocal reactions when tested by immunofluorescence in H9 cells infected with HIV. HIV antigen was not detected in any of the antibody-containing lots by the Abbott EIA for HIV antigen(s) (table). One of the manufacturers told us that reverse transcriptase could not be detected in cultures of H9 cells inoculated with their serum control lots. Nevertheless, as donors infected with HIV contributed to the pools from which the serum controls were prepared, these reagents should be regarded as contaminated with HIV and therefore potentially infectious for laboratory personnel.

Correction of plasma protein concentrations for haemodilution

My attention has been drawn to the fact that the formula used by my colleagues and me to adjust the concentrations and plasma proteins for changes in haematocrit in vivo does not apply in vitro. I propose an amended formula and show its validity for in vitro applications (table).

Addition of a volume (V) of isotonic saline to a sample of blood treated with a suitable anticoagulant in vitro will not change the total volume of the red cells present (RBC),
Effects on haematocrit and plasma protein concentration (albumin) of adding saline to a sample of blood

<table>
<thead>
<tr>
<th>Starting values</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Volume saline added (V) ml</td>
</tr>
<tr>
<td>2 Blood volume (BV) ml</td>
</tr>
<tr>
<td>3 Red cell volume (RBC) ml</td>
</tr>
</tbody>
</table>

The volumes could be ml or litres, with the corresponding protein content mg or g and the concentration could be expressed as mg/ml or g/l without changing numerical values.

Applying equation 12 to correct observed concentrations of plasma proteins to "O" time (for example, to values prior to cardiopulmonary bypass) would be likely to overcorrect if the time intervals between the "O" and subsequent samples were more than six hours because of the rapid exchange of most plasma proteins between the circulating blood and the tissue spaces. This has been observed after haemorrhage. The original formula, which is inappropriate in vitro is likely to have led to corrections fairly close to those correct in vivo, and which could otherwise be obtained using a computer model, requiring several assumptions. The above formula could be used to obtain rough estimates of the amounts of protein "added" to plasma in vivo by exchange from the tissues and the rate of the exchange.

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References

Book reviews


This comprehensive account of Hodgkin’s disease by an international group of distinguished investigators is particularly welcome at a time when considerable progress is being made towards understanding the nature of this enigmatic neoplasm. The scope of the book is impressive, ranging from topics as diverse as the biology of the Reed-Sternberg cell to the psychosocial effects the disease has on its victims. As might be expected advances in treatment, and somewhat ironically the problems generated by the very success of that treatment, receive particular emphasis, but those whose interests lie in epidemiology or special investigative techniques will not be disappointed. In this regard the central importance of accurate pathological diagnosis is properly recognised, even though some recent developments of interest relating to immunohistochemistry and gene

and the resulting haematocrit is easily calculated from the initial volume of the blood sample (BVv), the volume of saline added, and the initial haematocrit (Hcto). This is illustrated in the top six rows of the table, in which the initial volume of the blood sample is taken to be 5 ml and the haematocrit 0-4. Similarly, if saline is the diluting fluid the protein content of the diluted plasma remains constant, although the concentration falls with increasing dilution. This is illustrated in rows 8, 9, and 10 of the table.

The haematocrit is defined by Hct = RBC/BV, so that Hcto = RBC/BVv and Hctn = RBC/BVn, from which it follows that Hctn/Hcto = BVv/BVn (noting that BVv is the blood volume corresponding to Hctn). In a similar way the protein content (P) of the samples is unchanged, and if initial and diluted protein concentrations and plasma volumes are represented by Pco, Pcn, PVo and PVn, respectively, P = Pcn. PVv = Pcn. PVn; so that Pco = Pcn. PVv/PVn; noting that Pco is the desired "corrected" protein concentration to be calculated from the available measurements of the haematocrits and Pcn.

The ratio of the plasma volumes can be obtained from the haematocrits using PV = BV–RBC and BV = RBC/Hct; so that PV = RBC/Hct – RBC = RBC (1 – Hct/Hct)

Substituting the appropriate expressions for the plasma volumes in the above equation allows RBC to cancel out and gives:

Pco = Pcn
LHctn
Hcto
(1 – Hct)
Hctn
Hcto (1 – Hct)
Hctn
Hcto (1 – Hct)
Hctn
Hcto (1 – Hct)
Hctn
Hcto (1 – Hct)
Hctn
Hcto (1 – Hct)
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