Technical method

8 After five seconds' staining they are removed and briefly passed through water, 60% acetone, and acetone and then air dried.

9 The slide may be viewed using bright field transmitted light when titanium-containing areas appear yellow. Better visualization is achieved using reflected light with dark field illumination.

Results

Immersion of the section in sodium metasilicate ensures good reproduction of the original tissue morphology as this forms insoluble silicium dioxide when heated. The initial heating period converts any metal present to its oxide which in turn is converted to the sulphate by the sulphuric acid. Titanyl sulphate then forms a yellow precipitate with the tannic acid solution.

The series of controls showed that:
1 This sequence of chemical changes takes place with titanium salts in vitro and no interference is experienced when aluminium, cobalt, copper, chromium, iron or nickel are present.
2 A yellow precipitate is not produced by any of these other salts using the same technique.
3 Titanium may be identified in animal tissue after impregnation of the tissue with titanium salts using this technique.

Figure 2 shows a section of human tissue obtained from the location of a titanium implant that has been stained with haematoxylin and eosin while fig 3 shows a further section stained using the technique described. Figures 4 and 5 show sections of human tissue containing graphite that have been stained by the same methods, illustrating the similarity of the two haematoxylin and eosin stained sections but the differences between titanium- and non-titanium-bearing sections when stained with tannic acid.

Our thanks are due to Mr G. Abbott for construction of the microincineration furnace, to Miss E. Mort for technical assistance, to Mr J. S. Bailie and the Department of Medical Illustration for the illustrations, and to Dr G. Meachim for advice and provision of the graphite-containing material.

References


Letters to the Editor

No mouse model for H. influenzae infection

In a recent series of experiments investigating the toxic components of H. influenzae type b, an animal model was required for the demonstration of possible in vivo effects.

There are several reports on the effects of injecting or inoculating rabbits and rats with H. influenzae (Schneerson and Robbins, 1971; Smith et al, 1973; Moxon et al, 1974). As in humans, rabbits show an age-dependent susceptibility (with a maximum susceptibility at three weeks), which is explained by the level of serum anti-type b antibody.

Rats also show an age-dependent susceptibility to H. influenzae (only young rats are susceptible) but this does not appear to be influenced by the level of serum antibody (Smith et al, 1973).

As the mouse would have been the most suitable animal from the point of view of availability and general convenience it was decided to investigate the possibilities of a mouse model for H. influenzae infections.

There are two reports of infection in young mice following injection of H. influenzae: one describes injection of a mucin-enhanced suspension (Alexander et al, 1944) and the other injection of infected CSF from children suffering from meningitis (Wollstein, 1911). No report of the effect of injection of a known viable bacterial count of H. influenzae in mice was found.

We carried out two experiments on 11 litters of strain CD-I mice (Charles Rivers (UK) Ltd) from 0 to 10 days old each litter containing between 8 and 15 mice. H. influenzae type b (NCTC 8467) was grown in Brain Heart Infusion (fortified with haemin and NAD), washed, counted, and resuspended in PBS and injected one hour later. Each mouse was injected (in the first experiment subcutaneously and in the second intraperitoneally) with a 0·05 ml suspension containing 10^7 viable organisms. The mice never looked ill and no deaths occurred in either series of experiments. The animals were killed at five days, the blood expressed from the head was cultured, and H. influenzae was isolated from two mice only, both in the intraperitoneal injection experiment—one from the newborn group and one from the one-day group. In a third experiment a massive
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of the 11 patients among the group of 23 who had an FDP level higher than
16 mg/l within 48 hours of admission three (27·7\%) died. There was only one
death among the remaining 12 patients with an FDP level lower than 16 mg/l (8·3\%). These mortality percentages are similar to those reported by others for patients with high FDP levels—namely, 26\% (Almer et al., 1972a), 52\% (Baele et al., 1973), and 22\% (Okuno and Nelson, 1974). However, when considering the maximum FDP level during the whole of a patient’s stay in hospital the mortality percentages were different. Of 21 patients with a maximum FDP level higher than 16 mg/l three (13·6\%) died, and of 17 patients whose FDP level was never above 16 mg/l two (11·8\%) died. Thus, an increase in the FDP level seems to be of real prognostic value only when it occurs in the first 24 to 48 hours.

The results of a study of coincidental serum levels of the myocardial enzymes CPK and LDH, and of FDP are more interesting. Out of eight patients with raised FDP levels (> 16 mg/l) and levels of CPK > 400 U/l and of LDH, > 500 U/l three (37·5\%) died (see table). But out of three patients with raised FDP levels and low enzyme levels none died. Out of seven patients with a high level of one or both enzymes and a low FDP level only one (16·6\%) died. Finally, out of six patients with low levels of both FDP and enzymes none died.

These findings suggest that FDP and ME levels are of more prognostic value

<table>
<thead>
<tr>
<th>Serum Level</th>
<th>No. of Patients</th>
<th>No. died</th>
<th>Mortality Rate (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>FDP high</td>
<td>8</td>
<td>3</td>
<td>37·5</td>
</tr>
<tr>
<td>ME low</td>
<td>6</td>
<td>1</td>
<td>16·6</td>
</tr>
<tr>
<td>FDP high</td>
<td>6</td>
<td>1</td>
<td>16·6</td>
</tr>
<tr>
<td>ME low</td>
<td>23</td>
<td>4</td>
<td>17·4</td>
</tr>
</tbody>
</table>

Table Mortality in 23 patients with acute myocardial infarction correlated with serum FDP levels in the first 48 hours and maximum serum ME (CPK and/or LDH) levels during the patients’ stay in hospital

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