Slide coagulase positive, tube coagulase negative *Staphylococcus aureus*

We read with interest the letter by Smyth et al and wish to report a similar organism isolated recently at our hospital. The organism was grown in six blood culture bottles from a 14 year old boy with hypertrophic cardiomyopathy. An extensive intramyocardial abscess was seen on echocardiography. The isolate was rapidly and unequivocally positive using fibrinogen sensitised sheep erythrocytes in a slide test to detect clumping factor (Staphyslide-Test, BioMérieux, France). Tube coagulase testing was repeatedly negative. The isolate was strongly DNase positive and produced acid aerobically from maltose, trehalose, mannotol, mannos and sucrose but not from xylose, cellobiose, nor raffinose. Nitrate was reduced, acetoin was produced, and the organism was sensitive to novobiocin. These tests confirmed the identification as *Staphylococcus aureus*.

We are at present changing our laboratory procedure from routinely performing tube coagulase testing in all staphylococci to the use of "Staphyslide" to test for *S aureus* with tube coagulase as an additional test for "Staphyslide" negative colonies. From over 300 tests run in parallel we have yet to find a false positive or a false negative slide test for a methicillin sensitive *S aureus*.

In the light of the experience with these cases, ours, and that of Smyth et al, laboratory staff should be aware of the rare occurrence of false negative tube coagulase tests. We consider "Staphyslide" to be a sensitive and specific test for speciation of *S aureus* and suggest that its use should be considered for early identification of *S aureus*.

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Hair "follicle" in tonsil

The description of a hair "follicle" growing in the palatine tonsil is a misinterpretation. I suggest that what the authors were viewing was flattened, normal tonsillar crypt epithelium closely adherent to a foreign fragment, possibly an ingested hair shaft. I, too, have seen hair shaft-like particles in the tonsil. These were, like the reported case, separated from the crypt epithelium by an amorphous layer—eosinophilic in my original (figure). A semblance of neither the glassy membrane nor the connective tissue sheath, which are characteristic of hair follicles, cannot, on the other hand, be identified in the tonsillar structure shown in the letter or in my own material.

Contrary to the statement in the letter of Hasleton et al, there is indeed a reference to hair follicles growing in the tonsil provided in my book. This is with regard to hairy polyp, or teratoid tumour, a neoplasm which usually occurs in the pharyngeal tonsil, but which may be sometimes seen in the soft palate adjacent to the palatine tonsil. It is highly unlikely, however, that an isolated hair follicle will ever be found in any part of the tonsil.

Sebaceous glands opening directly on to the oral mucosa—Fordyce spots—are common in the oral cavity, but they are never associated with hair follicles.

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References

Dr Hasleton et al comment:

We thank Professor Michaels for his comments, but we would like to make several points. Hair follicles are not mentioned in the

Figure Palatine tonsil from 20 year old man with recurrent sore throat. Three oval, foreign particles resembling hair shafts are seen in this field. Two of them are surrounded by an oval layer—eosinophilic in the original—external to which are flattened cells (arrow) which are the specialised stratified squamous epithelial cells of the crypt.
normal anatomy section of the palatine tonsil in his book, and certainly we did not regard this a teratoid tumour.

This hair, we think, is unlikely to have been ingested because there is a well defined follicle adjacent to normal tonsillar tissue. Ingested hairs would be expected to have associated fibrosis and giant cells—not seen in our case. Levels had been cut from our lesion and no fibrosis or giant cells were identified.

This case illustrates the beauty of histopathology—one photograph can give rise to several interpretations and perhaps we are both within a “hair’s breath” of the truth.

**Dipstick urinalysis for bacteriuria**

We were interested to read the description by Doran and Kensit of the use of the Clinitec 200 to predict the presence of bacteriuria. This apparatus has been evaluated recently in a similar study in our laboratory.

A total of 1085 urine samples from hospital and community patients were examined. Each sample was tested with a multiple reagent strip for blood, protein, nitrite and leucocyte esterase and the results read semiautomatically by a Clinitec 200 reflectance photometer (Ams Laboratory, Slough, Buckinghamshire, England). One or more positive tests was scored as a positive result. The urine samples were then cultured semiquantitatively on cysteine lactose-deficient (CLED) agar and counts of $\geq 10^5$ organisms/ml taken as an indication of significant bacteriuria.

Of the 1085 samples, 726 (67%) were negative by the reagent strips but 18 (2%) of these subsequently grew significant numbers of bacteria on culture. Of the remaining 359 (33%), which were positive by the reagent strip tests, 120 (11%) were false positive results. The indices using the formulas described by Krieg et al$^2$ are set out in the table.

Despite an apparently high degree of success in excluding bacteriuria, it must be remembered that in our study there were still 18 false negative results, accounting for 7% of all samples with culturally confirmed bacteriuria. This figure, which is similar to the 10% reported by Doran and Kensit,$^1$ is substantial. Inevitably patients with urinary tract infection would fail to have the diagnosis made (or confirmed) and information about the antimicrobial sensitivity of the causal organism would never be available.

Because there were many fewer false positive results in our study, there was a considerable increase in the specificity and the predictive value for a positive reagent test. The specificity we report was also higher than the 38% observed by Lowe.$^3$ It is interesting that our result is in keeping with the 76% specificity of manual dipstick urinalysis reported in a recent comparative evaluation of screening methods for bacteriuria in this journal.$^4$ In that study, however, the relatively non-specific protein test was excluded. The reasons for these discrepancies are not clear, but at a practical level would directly influence the number of urine samples unnecessarily cultured.

The method is not attractive unless the investment in time and expense of using the Clinitec 200 is more than compensated by a reduction in the work of culturing negative specimens. A further problem is that all 18 patients are likely to have at least one dipstick urinalysis carried out on the ward and consequently the use of the Clinitec 200 in the hospital laboratory will duplicate tests already done—although they may be carried out more accurately.

There is not a clear cut case for the use of this method as a routine screening procedure in microbiology departments. It disappoints because of inaccurate results—negative results that lead to a missed diagnosis and positive ones that fail to reduce the workload. There is also no indication of a substantial financial saving. Perhaps the method could find a niche in screening selected patient groups such as pregnant women and children for asymptomatic bacteriuria.

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**Table** Comparison of different indices used to estimate value of Clinitec 200 as predictor of bacteriuria in two studies

<table>
<thead>
<tr>
<th></th>
<th>This study (n = 1085)</th>
<th>Doran and Kensit study (n = 669)</th>
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<tbody>
<tr>
<td>Sensitivity (%)</td>
<td>93</td>
<td>91</td>
</tr>
<tr>
<td>Specificity (%)</td>
<td>85</td>
<td>38</td>
</tr>
<tr>
<td>Predictive value for positive result (%)</td>
<td>66</td>
<td>26</td>
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<tr>
<td>Predictive value for negative result (%)</td>
<td>97</td>
<td>95</td>
</tr>
</tbody>
</table>

**References**

2. Krieg AF, Gambino R, Galen RS. Why are clinical tests performed? When are they valid? JAMA 1975;233:76-8.

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**Matters arising**

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**Book reviews**


This volume is the fourth of a new series entitled Current Problems in Tumour Pathology—the Pathobiology of Malignant Disease. No doubt the editors of different volumes in the series will interpret the term “pathobiology” differently. In this volume, the two editors, Dr Habeshaw and Professor Launder, have assembled a group of distinguished contributors from a variety of different disciplines, each of whom has written an essay on a topic related to some aspect of malignant lymphoma in which the author has particular expertise. The subjects range from epidemiology to the cellular origin of Hodgkin's disease and from cytogenticics to diagnosis, staging, and management. Although several of the individual contributions are outstanding, it is not easy to see for what class of reader the book as a whole is intended. The volume is beautifully produced and the illustrations are of a high quality but the text is unfortunately marred by many typographical errors.

AG STANSFELD


This book gives a comprehensive account of the vulva and its pathology starting with the background embryology, anatomy, and physiology, ranging through the various clinical and pathological aspects of vulval disease, and even including historical and psychological considerations. Everything from nappy rash and the psychological effects of rape to techniques of DNA hybridisation is here. While it may be admirable to have such an all-inclusive account of this anatomical area, it does beg...