ing January–December and April to December 1989 respectively. All were screened routinely for *Neisseria gonorrhoeae*, syphilis and chlamydia (for which we used an ELISA technique (Boo! Celltech)). In females, cultures and wet films for *Candida albicans* and *Trichomonas vaginalis* were carried out. Anaerobic vaginosis was diagnosed in women who had symptoms of malodorous vaginal discharge, and clue cells found on microscopy.

In the females we found some similarities with Griffiths, namely a high prevalence of *Candida albicans*, a low prevalence of *Trichomonas vaginalis* (1 female), and *Gonorrhoea* (1 female). There was, however, a high prevalence of anaerobic vaginosis in our patients. It was felt that for this diagnosis to be made, the criteria should have included vaginal pH sampling with a pH > 5 and a positive amine test. This may have resulted in a smaller percentage of cases of anaerobic vaginosis in our study. We found 12% of women to have *Chlamydia trachomatis* present. In 79% of males, no other infection was found, but chlamydia or non-specific urethritis was diagnosed and treated in 14% (table).

We felt that the studies of Longhurst and Turner *et al* do not accurately represent the population of "young sexually active women in general". Both groups contain women who we could consider to be at high risk for sexually transmitted disease. The social and demographic features of Longhurst’s group were biased towards patients at the extremes of social class. Many were underprivileged, 20% lived in overcrowded conditions, 17% belonged to minority ethnic groups and 13% were unemployed. Eight per cent of the consultations were with temporary residents, many of them students. This practice cannot be a sample of general practice as a whole.

Turner’s study selected out a group with abnormal cervical cytology, which warranted colposcopy. It is now recognised that wart virus infection plays an important role in causing abnormal cytology. Therefore the above group could also be a biased sample.

There are several reasons why we consider screening for other sexually transmitted diseases to be good practice within a genitourinary department. Criteria for screening include availability of simple, cheap tests, which are reliable and sensitive. Tests should detect conditions where pathogenicity is severe enough to warrant screening; and treatment should be easily available once detected. (Chlamydia and gonorrhoea certainly fulfil these criteria.)

Detection of genital warts provides an opportunity to screen a high risk group of both women and men for sexually transmitted infection which may be asymptomatic, making selective screening difficult. Screening in genitourinary clinics, together with contact tracing is vital in containing the infection. The known link between presence of human papillomavirus and pre-cancerous changes of the cervix also reinforces the need to include cervical cytology in any routine screening of women presenting with genital warts.

Finally, Griffith’s message is to urge us not to ignore the risk of STDs in other women who are sexually active, but may not attend the genitourinary clinic. We would advocate full screening within the genitourinary clinics for all patients, including those with warts, and also all women deemed to be at high risk within the sexually active group. These could include those attending for colposcopy or those in socially deprived areas, but at present it would not be feasible to include all “young sexually active women in general” as the criteria for selection in such a group is not well defined.

### Table. Prevalence of lower genital tract infection in 127 women and 147 men presenting with warts

<table>
<thead>
<tr>
<th>Condition</th>
<th>Females (%)</th>
<th>Males (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Candida</em></td>
<td>20%</td>
<td>0%</td>
</tr>
<tr>
<td><em>Chlamydia trachomatis</em></td>
<td>12%</td>
<td>8%</td>
</tr>
<tr>
<td>Bacterial vaginosis</td>
<td>16%</td>
<td>0%</td>
</tr>
<tr>
<td>Non gonococcal urethritis</td>
<td>6%</td>
<td></td>
</tr>
<tr>
<td><em>Trichomonas</em></td>
<td>One case —</td>
<td></td>
</tr>
<tr>
<td><em>Gonorrhoea</em></td>
<td>One case —</td>
<td></td>
</tr>
<tr>
<td>Any of the above</td>
<td>43%</td>
<td>14%</td>
</tr>
<tr>
<td>Other conditions</td>
<td>3%</td>
<td>7%</td>
</tr>
<tr>
<td>requiring treatment</td>
<td>54%</td>
<td>79%</td>
</tr>
</tbody>
</table>

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**Detection of human papilloma virus DNA in semen from patients with intrameatal penile warts**

Two points arise from the recent paper by Green and colleagues. Fifteen samples from ten patients were tested and five samples were positive. Clearly at least one man must have produced more than one sample; were any two positives from the same man? Were findings consistent between samples?

Secondly the authors quote a paper by Levine and colleagues in support of their view that "Human papillomavirus (HPV) infections of the genital tract are sexually transmitted". Levine’s paper in fact demonstrated an association between "cervical condyloma" or CIN1 in women with the presence or past history of penile warts in their partners. No DNA hybridisation was carried out in either sex. That genital warts are sexually transmitted is long established; however, a recent editorial in this journal by the same author pointed out that individual HPV types have not been proved to be sexually transmitted. Although to some this may seem to be a reasonable conclusion, there is no evidence to support the assumption whereas evidence to the contrary exists.

Wickenden and colleagues showed evidence for the sexual transmission of HPV-6 and 11—but not types 16 and 18. In a study of the male partners of women shown to have cervical HPV infection, the majority of the males did not harbour the same virus types as their partners.

For successive authors to state that HPV infections—particularly with the so-called "high-risk" types 16 and 18—are sexually transmitted when no supporting evidence exists, seems to...
establish a false dogma, of the sort which has been described as “The Bellman’s fallacy”—whereby if a mistruth is repeated often enough it becomes an accepted fact.

The extent of our present knowledge does not allow us to state how certain HPV types are transmitted. Until we know the facts, speculation should remain just that and not be translated into possibly false dogma.

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Green et al reply,
Thank you for showing us Dr Griffiths’ letter. We received 15 specimens from 10 patients, five patients providing two specimens, five patients providing one specimen. Five specimens were positive for HPV DNA. The three stronger HPV-DNA positive specimens came from patients providing only one specimen, but we cannot say whether the two other positives came from one or two patients as two specimens lost their code numbers in transit.

Dr Griffiths’ second point concerns the evidence for the sexual transmission of genital HPV infection. Sexual transmission of genital warts is long established, and Dr Griffiths accepts the evidence of Wickendon and colleagues for the sexual transmission of HPV types 6 and 11 between couples with genital warts. Dr Wickendon does not suggest that only HPV types 6 and 11 are sexually transmitted, nor does his study provide “evidence to the contrary” that sexual transmission of other HPV types can occur. Careful analysis of Schneider’s study, referred to by Dr Griffiths, reveals that in the majority of cases where HPV was recovered from both partners the viral type was the same. Evidence for sexual transmission, the demonstration of infection of both partners, exists for HPV16 and HPV18 in the case of penile and cervical lesions.21


BOOK REVIEW


This small compact book has 15 chapters (with 18 contributors) covering most aspects of HIV infection, epidemiology, virology and immunology, clinical care (including counselling, obstetric and paediatric issues) and treatment. It is the sort of book, aimed at junior hospital doctors and medical students, that can be easily carried in a white coat pocket, although GPs, nurses and other paramedical staff will find it useful. Indeed I am exactly the sort of person the book is NOT aimed at, and for that reason I asked some of our junior doctors and nurses their opinion also, all of whom were extremely impressed—in fact I had difficulty in getting the book back on two occasions!

However, anyone with experience in the field of HIV infection may find the book frustrating in two main respects. Because of limited space the editor has chosen not to reference the text, giving only a list of “further reading” at the end of each chapter. Although I understand the motives for doing this, where certain perhaps controversial practices are quoted in detail, I think the source of the information should be clearly stated.

The other problem with the book is that it very much reflects the current practice at one centre, not clearly indicating where there are different schools of thought. For instance, in the chapter on neurological disease Carne and Harrison quote the treatment for cytomegalovirus infection as being “Ganciclovir 2.5–5 mg/kg tds for three weeks”. I think that most centres would feel this was an unnecessarily excessive dose for the majority of patients (5 mg/kg bd for 14 days often being sufficient) and may be toxic. The management of neutropenia, concomitant treatment with zidovudine and the indications for the alternative drug foscarnet are not mentioned although these are important practical issues. Similarly in the chapter covering the respiratory manifestations of AIDS it is stated that bronchoscopy need not be performed in a patient with the “typical clinical and radiographic presentation of PEP [PCP] who is hypoxaemic” and should only be performed if any deterioration occurs or if the patient fails to improve. No mention is made of the problem of multiple pathology in HIV associated pneumonia, or the fact that if a patient deteriorates he may become too sick to bronchoscope.

Many centres prefer a policy of performing a bronchoscopy early, and I think particularly in a book aimed at an inexperienced group of doctors in such cases where there is often no cut and dried answer, both sides of the argument should be aired.

These criticisms apart, there is no doubt that many people will find this an extremely useful book. It is clear, concise, covers an amazing amount of information for such a small volume and some of the chapters (for instance those by Quentin Sattentau on the Virology of AIDS, and Ian Weller on Treatment and Prevention) are really first rate. I am sure, particularly in view of its reasonable price, it will be widely read.

SM FORSTER
Detection of human papilloma virus DNA in semen from patients with intrameatal penile warts.

M Griffiths

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