228 Letters to the Editor

the treponemicidal level is achieved and maintained there is no evidence that higher levels are more effective against the treponeme.

Fishman⁴ showed, in the experimental animal, that treatment with probenecid in addition to penicillin in equimolecular amounts increased the level of penicillin in the brain by X1·9. This was accompanied by a rather greater increase of X2·9 in plasma, so that the ratio of brain to plasma penicillin decreased from $5\cdot4^{\circ}_{\circ}$ to $3\cdot5^{\circ}_{\circ}$.

Probenecid with penicillin or amoxycillin's should be as effective in the treatment of neurosyphilis, providing treponemicidal levels are produced and maintained, as the more complicated intravenous regimens. The matter deserves further examination.

ERIC MC DUNLOP*
SAFWAT S AL-EGAILY†
ELIZABETH T HOUANG†
*Retired from the Ambrose King Centre
(Whitechapel Clinic), The London Hospital,
(present address for correspondence:
26 Old School Lane, Milton,
Cambridge CB4 4BS, UK
†Huddersfield Royal Infirmary; formerly
Ambrose King Centre (Whitechapel Clinic),
The London Hospital
†Department of Medical Microbiology,
Queen Charlotte's and Chelsea Hospital,
formerly Department of Medical
Microbiology, The London Hospital

- 1 van der Valk PGM, Kraai EJ, van Vöorst Vader PC, Haaxma-Reiche H, Snijder JAM. Penicillin concentrations in cerebrospinal fluid (CSF) during repository treatment regimen for syphilis. Genitourin Med 1988;64:223-5.
- 2 Dunlop EMC, Al-Egaily SS, Houang ET. Production of treponemicidal concentration of penicillin in cerebrospinal fluid. Br Med J 1981; 283:646.
- 3 Dunlop EMC, Al-Egaily SS, Houang ET. Penicillin levels in blood and CSF achieved by treatment of syphilis. JAMA 1979;241:2538-40.
- 4 Fishman RA. Blood-brain and CSF barriers to penicillin and related organic acids. Arch Neurol 1966; 15:113-24.
- 5 Faber WR, Bos JD, Rietra PJGM, Fass H, van Eijk RVW. Treponemicidal levels of amoxicilin in cerebrospinal fluid after oral administration. Sex Transm Dis 1983;10:148-50.

Colposcopy in Teenagers

The incidence of pre-invasive squamous carcinoma of the cervix

Age distribution against cervical biopsy

Age	No	Cervical biopsy results				
		Normal or inflammatory	HPV histology	CIN 1	CIN 2	CIN 3
15	1		_	1		_
16	12	2	5	3	2	
17	15	2	5	6	1	1
18	32	5	14	9	4	_
19	36	11	15	9	1	_
Totals	96	20	39	28	8	1

among younger women is increasing steadily in England and Wales.12 It has been suggested that these lesions progress rapidly to invasive cancer in younger women.3 We screened, cytologically, colposcopically and histologically, 96 teenagers with genital warts for cervical intraepithelial neoplasia, whose mean age was 17.9 (range 15 to 19 years). Seventy (73%) of them were smokers and 69 (72%) of them used an oral contraceptive pill; only 15 (15.6%) used barrier methods. The majority of them were having regular sexual intercourse from the age of 16 and continued to have multiple sexual partners. Histological evidence of cervical intraepithelial neoplasia was found in 37 (38.5%) patients (table).

Epidemiological studies indicate that principal risk factors for cervical neoplasia are early engagement in sexual intercourse, multiple partners⁴ and cigarette smoking.⁵ Recent data have suggested that long term use of oral contraceptive increases the risk of cervical cancer.⁶ It has also been established that a close link exists between cervical intraepithelial neoplasia and human papilloma virus.⁷

The detection of a high incidence of cervical intraepithelial neoplasia among teenagers is rather alarming. Our study has shown that the majority of these teenagers have multiple risk factors for cervical neoplasia. We feel strongly that a colposcopic examination and regular follow-up is mandatory in these groups of patients. Our results support the argument that cervical screening should start from an earlier age than currently recommended.

K SIVAKUMAR AH DE SILVA R BASU ROY Department of Genito-Urinary Medicine Royal Victoria Hospital, Gloucester Road, Bournemouth BH7 6JF, UK

- 1 Roberts A. Cervical cytology in England and Wales 1965-80. Health Trends 1982;14:41-5.
- 2 Wolfendale MR, King S, Usherwood MM. Abnormal smears; are we in for an epidemic? Br Med J 1983;287: 526-8.
- 3 Patterson MEL, Peel KB, Joslin CAF. Cervical smear histories of 500 women with invasive cancer in Yorkshire. Br Med J 1984;289:896-8.
- 4 Kessler I. Venereal factors in human cervical cancer: evidence from marital cluster. Cancer 1977;39:
- 5 Harris RWC, Brinton LA, Cowdell RH, et al. Characteristics of women with carcinoma in situ of cervix uteri. Br J Cancer 1980;42:359-69.
- 6 Beral V, Hannaford P, Kay C. Oral contraceptive use and malignancies of the genital tract. Lancet 1988;ii:1331-4.
- 7 McCance DJ, Campion MJ, Clarkson PK, Chesters PM, Jenkins D, Singer A. Prevalence of human papillomavirus type 16 DNA. Sequence in cervical intraepithelial neoplasia and invasive carcinoma of cervix. Br J Obstet Gynaecol 1985;92:1101-5.

MATTERS ARISING

Genital warts and the need for screening

Griffiths et al reviewed 100 consecutive women with genital warts to determine the prevalence of associated lower genital tract infection. He concludes that screening all women with genital warts for other sexually transmitted diseases, whilst ignoring a similar risk in women without warts, may be inappropriate. In an attempt to assess the situation for ourselves, we looked at 127 consecutive women and 145 consecutive men, presenting dur-

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 (Boot 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 malodourous 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 nonspecific urethritis was diagnosed and treated in 14% (table).

We felt that the studies of Longhurst² and Turner et al³ 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

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

Condition	Females	Males
Candida	20%	0%
Chlamydia trachomatis	12%	8%
Bacterial vaginosis	16%	0%
Non gonococcal urethritis	_	6%
Trichomonas	One case	_
Gonorrhoea	One case	
Any of the above	43%	14%
Other conditions requiring treatment	3%	7%
No infection	54%	79%

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.⁴ 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.

> SC CRAWSHAW MV HARAN Stafford District General Hospital, Weston Road, Stafford ST16 3SA, UK

- 1 Griffiths M, et al. Genital warts and the need for screening. Genitourin Med 1989;65:399.
- 2 Longhurst LJ, Flower N, Thomas BJ, et al. A simple method for detection of Chlamydia Trachomatis infection in general practice. J R Coll Gen Pract 1987;37:255-6.
- 3 Turner MJ, White JO, Soutter WP. The male factor in cervical neoplasia. Contemp Rev Obstet Gynaecol 1988;1: 36-42
- 4 Young LS, Bevan IS, Johnson MA, et al. Polymerase: a new epidemiological tool for investigating cervical human papilloma virus infection. Br Med J 1989;298:14-18.

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 colleagues² in support "Human their view that 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;3 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 colleagues⁴ 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.⁵

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