Oral sex is not safe sex

To the Editor: In the last few months, since the outbreak of the acquired immunodeficiency syndrome epidemic, a number of my patients with typical nonspecific urethritis gave a history of having had fellatio performed. The results of urianlysis revealed white blood cells. On the basis of direct fluorescent antibody smear technique some showed positive findings for Chlamydia trachomatis. Because the sex partners of these patients (many of whom had had oral sex with either a prostitute or on “one-night stands”) had never come to my office until recently, it was impossible to draw a relationship until now. It seems that many people, including prostitutes in the New York area, now consider oral sex to be safe, or at least a safer form of sexual activity.

This letter is prompted by two recent patients who came to my office. One, a white, bisexual, 24-year-old man had nongonococcal urethritis; a direct fluorescent antibody smear was positive for C. trachomatis. His recent history included having had fellatio performed on him by only one male sex partner in the previous 4 weeks. Approximately 1 month before this sexual experience the patient was evaluated for venereal diseases because of a herpes simplex infection on his penis. Included in the evaluation was a fluorescent antibody technique smear from his urethra, which was negative for C. trachomatis. The male partner was asymptomatic and his throat, on physical examination, was normal; however, direct fluorescent antibody smear technique revealed a culture that was positive for C. trachomatis.

The second patient, a white, heterosexual, 45-year-old man, had a prostitute perform fellatio on him. When I saw the patient, he had a chancre on his glans penis. Serologic findings showed a titer of 1:256 and the result of fluorescent treponemal antibody, ABS was 4+ reactive.

In conclusion, we should be aware that fellatio and cunnilingus, although thought to be safe, are not, and that patients should be counseled not to engage in these practices without realizing the consequences of contracting venereal disease.

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REFERENCES

Pruritus confined to scapular or subscapular region? Occult Becker’s nevus with mild folliculitis

To the Editor: I read with great interest the report by Drs. Weber and Poulos (J AM AAD DERMATOL 1988;18:25-30). I have had three similar patients recently, each of whom had an area of pruritus confined to the scapular or subscapular region. On close inspection, slight hyperpigmentation and follicular prominence were noted. Treatment with 5% benzoyl peroxide lotion was effective in resolving the itching in all patients.

I suspect that notalgia paresthetica may represent an occult Becker’s nevus in which a mild folliculitis develops. The location, hyperpigmentation, and histopatholo-
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Skin tumors in the European PUVA Study.
Eight-year follow-up of 1643 patients treated with PUVA for psoriasis

To the Editor: The paper by Henseler et al. (J Am Acad Dermatol 1987;16:108-16) appears to offer some assurance to European dermatologists that treatment with psoralens plus ultraviolet A (PUVA) is not as carcinogenic as may be suggested by the 16-center study in the United States. Although a positive association is demonstrated between the incidence of tumor and cumulative dose of PUVA, the authors point out in Table V of their article that many of the 34 patients who acquired either basal cell or squamous cell carcinoma also had been exposed to other potentially carcinogenic substances (notably arsenic) before receiving PUVA. If, however, many of the remaining 1609 patients had also been exposed to arsenic, then arsenic would be discounted as the culprit. Inasmuch as the authors do not specifically address this question, it is somewhat difficult to draw conclusions. These figures reveal an estimated tumor incidence of 1.5% in the low-dose group and 11.1% in the high-dose group.

To test whether the dose of PUVA is independently associated with tumor incidence after allowing for prior exposure to arsenic, the authors should have divided their data: (1) patients exposed to arsenic and (2) patients not so exposed, as, for example, Table II here.

Unfortunately we still need to know how many of the 186 patients who were exposed to arsenic but who did not have tumors had received a low cumulative dose and how many had received a high cumulative dose of PUVA. We also need the same information concerning the 911 persons who were not exposed to arsenic and who did not develop tumors. With this information, incidence rates of tumors could be calculated for (1) those exposed to arsenic and receiving a high PUVA dose, (2) those exposed to arsenic and receiving a low PUVA dose, (3) those not exposed to arsenic but receiving a high PUVA dose, and (4) those not exposed to arsenic and receiving a low PUVA dose. Thus the "true" effect of PUVA would be assessed by comparing 1 with 2, and 3 with 4. The analysis could be performed with the use of a Mantel-Haenszel test for combining information from 2 x 2 tables.

Table I. Acquiring of squamous/basal tumor according to dose of PUVA

<table>
<thead>
<tr>
<th>PUVA dose</th>
<th>Tumor</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
</tr>
<tr>
<td>Low</td>
<td>1529*</td>
</tr>
<tr>
<td>High</td>
<td>80*</td>
</tr>
<tr>
<td>Totals</td>
<td>1609</td>
</tr>
</tbody>
</table>

*Estimated from Fig. 2, B, (Henseler et al.)
†From Table V (Henseler et al.)

Table II. Subdivision of data according to arsenic exposure

<table>
<thead>
<tr>
<th>PUVA dose</th>
<th>Arsenic</th>
<th>No arsenic</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Tumor</td>
<td>Tumor</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Low</td>
<td>18</td>
<td>4</td>
</tr>
<tr>
<td>High</td>
<td>7</td>
<td>2</td>
</tr>
<tr>
<td>Totals</td>
<td>186</td>
<td>25 211 911</td>
</tr>
</tbody>
</table>

From Tables IV and V (Henseler et al.)

It would surely have been possible for the authors to produce the necessary extra data, without which the reader cannot verify the conclusions made. Of course, allowing for exposure to arsenic does not make the low and high dose groups comparable in every respect. Many other variables also may be related to tumor incidence, many of which are unobservable. Follow-up of patients with psoriasis who are enrolled in randomized trials involving PUVA therapy would be the ideal. In the meantime the European PUVA Study has some useful data to offer. Perhaps the authors could complete their analysis for us.

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Reply

To the Editor: I thank Dr. Morris for his interest in our paper. We regret his impression about a need for further information. In response, we take the opportunity to provide some additional data to clarify the shortcomings mentioned by Dr. Morris.

Completion of the tables calculated by Dr. Morris (number of patients with low/high dose in Table 1, 1527/82; in Table II, 172/15; and in Table III, 845/36)