All the spectinomycin resistant *N gonorrhoeae* required proline for growth and carried the 2·6 and 24·5 megadalton plasmids. The penicillinase producers also carried the 4·4 megadalton plasmid. All strains showed high level resistance to spectinomycin (⩾512 mg/l). Two of these were not detected on initial screening with a 100 μg spectinomycin disc, and the other resistant strains grew up to the disc. Serological studies showed that all spectinomycin resistant *N gonorrhoeae* tested were indistinguishable. The serovar was Bacjk.

Despite the biological similarity among the strains they were isolated over a long period, and, although three contact pairs were seen, no clear overall epidemiological pattern was evident.

Clinical failure with spectinomycin that could not be accounted for by spectinomycin resistance occurred in 5% of patients, a figure compatible with other treatment regimens employed at the clinic.[9]

Discussion

The change from oral treatment to intramuscular spectinomycin did not prove to be a major problem, and by September 1983 some 85% of heterosexual patients with gonorrhoea were receiving spectinomycin. In common with others we have isolated few examples of penicillinase producing *N gonorrhoeae* (14/588) from male homosexuals[10] and consequently ampicillin and probenecid remained first line treatment for these patients. Some 2000 patients received spectinomycin in 1983.

In Los Angeles the introduction of spectinomycin combined with intensive epidemiological back up had a spectacular effect in reducing the prevalence of infections with penicillinase producing *N gonorrhoeae*.[8] We have seen no such effect, but there are important differences between the two settings. Firstly, the Praed Street Clinic is only one of many clinics in London for sexually transmitted diseases. Patients have ready access to all public clinics and can and do go from one to another. In this setting a change in treatment in one clinic, albeit the largest one, can have only a limited effect. Secondly, we do not have the financial resources to carry out intensive epidemiological work on the scale done in Los Angeles. We believe that this aspect is at least as important as the introduction of a penicillinase stable antibiotic.

If spectinomycin has not caused a dramatic fall in the prevalence of penicillinase producing *N gonorrhoeae*, has it prevented a dramatic rise? The increase in numbers of penicillinase producing *N gonorrhoeae* isolated yearly did slow down in 1983 after the dramatic rise during the previous five years. A similar pattern, however, was evident in the figures for penicillinase producing *N gonorrhoeae* isolated in England and Wales. During July to December 1983 there were 599 isolates of penicillinase producing *N gonorrhoeae* reported to the Communicable Disease Surveillance Centre. This compares with 624 in the preceding six months and 613 during July to December 1982.[11] It will be several months, however, before we know whether the same is true world wide.

Spectinomycin resistance in both penicillinase producing and non-penicillinase-producing gonococci has been a worrying development, but despite sporadic reports in Britain and a cluster of cases in Korea there is no evidence of dissemination of strains world wide. Although our early isolates of penicillinase producing *N gonorrhoeae* were all directly linked to spectinomycin treatment, we have since seen the apparent transmission of these organisms. Microbiological evidence suggests that we are dealing with a single clone of organisms, but we lack the epidemiological information to link them. Apart from possession of the 4·4 megadalton plasmid coding for penicillinase production, all the spectinomycin resistant *N gonorrhoeae* are so similar that they may well have a common source. This might be either a strain of penicillinase producing *N gonorrhoeae* that has lost its 4·4 plasmid or a strain of non-penicillinase-producing *N gonorrhoeae* that has gained it. The serological evidence also supports a common source. It is also possible, however—though highly unlikely—that we are observing a series of unconnected events.

We changed to spectinomycin as first line treatment using the principle suggested by McCutchan *et al* and McCormack that this should be done if the prevalence of penicillinase producing *N gonorrhoeae* rose above 5%.[3,4] Spectinomycin when used as first line treatment successfully cured 95% of all gonorrhoea at the Praed Street Clinic. Continued treatment with ampicillin and probenecid would have necessitated further treatment for all infections with penicillinase producing *N gonorrhoeae* (8·7%) together with an additional 5% of non-penicillinase-producing *N gonorrhoeae* infections (unpublished data). This overall failure rate of at least 14% justifies our policy of continuing to use a penicillinase stable drug, although these are some six times more expensive than ampicillin and probenecid. We do not believe, however, that the introduction of such a drug into a single clinic in a large metropolitan area such as London, served by many clinics, can influence the prevalence of penicillinase producing *N gonorrhoeae* either in that clinic population or in the population at large. Therefore, given these special circumstances in London, we must question the relevance of the 5% level.

Recently Thin *et al* commented that a penicillinase stable cephalosporin such as cefotaxime was preferable to spectinomycin for the treatment of infections caused by penicillinase producing *N gonorrhoeae*.[12] They based this on the emergence of spectinomycin resistance and on two clinical failures of spectinomycin that occurred despite in vitro sensitivity. Our overall clinical failure rate of 5% with spectinomycin is comparable with other regimens for gonorrhoea. Any resistance of *N gonorrhoeae* is worrying and needs careful monitoring. To date, however, we do not think that spectinomycin resistance among strains of *N gonorrhoeae* is a major clinical problem either at St Mary's Hospital or elsewhere in Britain. If the routine use of spectinomycin is not accompanied by careful follow up and screening of all gonococcal isolates, once resistance has occurred it could build up to serious levels very rapidly.

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Correction

Combined oral and nasal beclomethasone dipropionate in children with atopic eczema: a randomised controlled trial

In this paper by Dr R J Heddle *et al* (15 September, p 651) dipropionate was spelt incorrectly throughout.