Carriers of meningococci among staff from department in which meningococcal disease was present and from control departments

<table>
<thead>
<tr>
<th>No of staff</th>
<th>No (%) of carriers</th>
<th>Groups of meningococci</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haematology department:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bedside contact with index case</td>
<td>68</td>
<td>11 (16)</td>
</tr>
<tr>
<td>No contact</td>
<td>115</td>
<td>9 (8)</td>
</tr>
<tr>
<td>Infectious diseases and rheumatology departments</td>
<td>247</td>
<td>20 (8)</td>
</tr>
<tr>
<td>Total</td>
<td>430</td>
<td>40 (9)</td>
</tr>
</tbody>
</table>

were taken from all carriers three days after the end of treatment with rifampicin showed that meningococci were no longer carried.

Comment

Strains of meningococci carried in the general population usually belong to non-virulent serological groups and types, even during an epidemic, and hence the ratio of attacks to carriers is low; in Denmark in 1987 it was 1:17 000 (297 cases in a population of 5·1 million). If the secondary attack rate among household contacts of an index case of meningococcal disease is 500-800 times that in the general population1 then the estimated risk is 2·9-4·8%. Three of seven carriers identified in our study developed the disease, giving an attack rate of 43% (95% confidence interval 7% to 82%); consequently we gave chemoprophylaxis to household contacts of carriers.

Patients with lower respiratory tract infection from whom N meningitidis is isolated most commonly suffer from chronic pulmonary disease.2 The relative risk of secondary meningococcal disease among people in their vicinity is unknown. We therefore suggest that until more evidence has accumulated chemoprophylaxis should be considered for patients in the same room as a patient with respiratory tract infection caused by virulent meningococci of serogroup C:2a and for staff in contact with such patients.

Does eradication of meningococcal carriage in household contacts prevent secondary cases of meningococcal disease?

James M Stuart, Keith A V Cartwright, Priscilla M Robinson, Norman D Noah

From 1 October 1981 to 31 March 1988, 109 cases of meningococcal disease, mainly due to group B type 15 subtype 16 strains resistant to sulphamidine, were recorded in Gloucester Health District, six of them occurring among 309 household contacts of index patients. Two cases occurred 12 and 36 hours after admission of the index patient and before chemoprophylaxis was given. The four others occurred 28, 107, 147, and 156 days after the index cases. All household contacts and three of the four index patients had received rifampicin for two days as currently recommended,1 and postnasal swabs were negative after treatment.

Effectiveness in eradicating nasopharyngeal carriage of meningococci is considered an appropriate criterion for selecting chemoprophylactic agents for meningococcal disease.2 As most previous studies have examined short term effectiveness3 and as four secondary cases in this outbreak occurred up to five months after prophylaxis we examined whether carriage of outbreak strains was persistently reduced after rifampicin was given.

Subjects, methods, and results

During a community survey of 6234 people in November 1986,4 79 nasopharyngeal carriers of outbreak strains were identified. In December after a second postnasal swab 50 carriers received rifampicin (600 mg twice daily for adults, 10 mg/kg twice daily for children) for two days, and 29 declined treatment. Thirty three (66%) in the treated and 18 (62%) in the untreated groups were still carrying outbreak strains (table). In January 1987 only one of the treated group had a positive postnasal swab compared with 13 of the untreated group (table). If the natural rate of loss (28%)

<table>
<thead>
<tr>
<th>Time when swab taken</th>
<th>At time of treatment (December 1986)</th>
<th>After one month</th>
<th>After five months</th>
<th>After 11 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rifampicin None</td>
<td>33/18 (100)</td>
<td>1/3 (3)</td>
<td>0/3 (0)</td>
<td>0/3 (0)</td>
</tr>
<tr>
<td></td>
<td>18/18 (100)</td>
<td>1/18 (7)</td>
<td>0/18 (0)</td>
<td>0/18 (0)</td>
</tr>
</tbody>
</table>

*Patient received second course of rifampicin and swab was subsequently negative.

had applied to the treated group then the expected number of carriers would have been 24. Thus the effectiveness of rifampicin in eradicating carriage was 96% (23/24, 95% confidence interval 88 to 100%). In May and November two more patients in the treated group had positive postnasal swabs, and rates of carriage continued to fall slowly in the untreated group.

Comment

Support for the practice of prescribing antibiotics such as rifampicin to household contacts of patients with meningococcal disease2,5 comes from a retrospective study of rates of secondary attack with limited follow up.2 Unlike the policy of mass prophylaxis in military communities,1 it has never been evaluated by controlled trial.

Our study showed that persistent eradication of nasopharyngeal carriage of meningococci can be achieved by giving rifampicin for two days. Despite the apparently low rate of reacquisition four cases occurred one to five months after prophylaxis and the rate of


(Accepted 21 December 1988)
secondary attack was higher than expected. House-  
hold contacts of a patient had a relative risk of infection  
750 times that of other people in the health district (1-94/0-0026). Possibly the rate of secondary attack  
would have been higher still without prophylaxis.  
Possibly, too, prophylaxis did not reduce rates of  
attack but merely delayed the onset of secondary  
cases in the family; the three patients whose families  
received "correct" prophylaxis developed the disease  
more than three months later. Successful eradication  
of carriage within the household cannot prevent outbreak  
strains re-entering the family; the interval depends on  
the prevalence and rate of transmission of outbreak  
strains in the local population.  

Whether or not prophylaxis has been given the  
general practitioner and members of the family should  
remain vigilant after a case of meningococcal disease. A  
randomised controlled trial is needed to test the  
hypothesis that eradicating meningococcal carriage in  

household contacts prevents further cases of meningo-  
occal disease.

This study was supported financially by the Department  
of Health and Social Security. We thank Drs Dennis Jones and  
Stephen Palmer for their constructive comments on this paper  
and the staff of the public health laboratories in Gloucester  
and Manchester for technical help.

1 Public Health Laboratory Service Communicable Disease  

2 Centers for Disease Control. Analysis of endemic meningococcal  

3 Broome CJ. The carrier state: Neisseria meningitidis. J Am Microbiol Chemother  

4 Cartwright KAY, Stuart JM, Jones DM, Nash ND. The Stonehouse survey:  
Nasopharyngeal carriage of meningococci and Neisseria lactamica. Epidemol Infect  

5 De Wals P, Hertoghe L, Borette-Grimme I, et al. Meningococcal disease in  
Belgium. Secondary attack rate among household, day care nursery and pre-  

(Accepted 22 December 1988)

Nicotine absorption and dependence in an over the  
counter aid to stopping smoking

Michael Belcher, Martin J Jarvis,  
Gay Sutherland

The importance of nicotine dependence in cigarette  
smoking and as a deterrent to stopping is receiving  
increasing recognition. This stems partly from the use  
of nicotine replacement methods to treat dependent  
smokers. Nicotine chewing gum, available only on  
prescription, is the only nicotine replacement treat-  
ment that is licensed in the United Kingdom. We  
investigated the absorption of nicotine from an over the  
counter aid to stopping smoking (Stoppers; Leo  
Laboratories).

Case report and study

A 38 year old man who had smoked hand rolled  
cigarettes for over 20 years stopped smoking with the  
help of nicotine chewing gum (Nicorette) 2 mg from  
his general practitioner. After he had used 15 pieces  
for two months he broke a tooth while chewing. He  
then started taking Stoppers, describing the transition  
as effortless, and was soon taking 30-60 lozenges a day.  
He contacted our clinic after a failed attempt to stop  
them after two years' use. Stopping taking Stoppers  
resulted in his feeling irritable, ill at ease, unable to  
concentrate, depressed, and hungrier than usual.  
These symptoms of withdrawal from tobacco were  
rapidly relieved when he resumed taking Stoppers  
after four days' abstinence. We took a blood sample  
just after he had finished one lozenge, after a total of 20  
on the day. Plasma nicotine and cotinine concentra-  
tions were 12.9 μg/l and 415 μg/l respectively. An  
expired air carbon monoxide concentration of 3 ppm  
confirmed that he had stopped smoking.

He bought his lozenges in bulk from the manufac-  
turer, partly for economic reasons as a discount was  
offered and partly because of anxiety about running  
out. He also believed that these lozenges were stronger  
and more satisfying than lozenges purchased from  
pharmacists.

We tested lozenges obtained from local pharmacists  
and directly from the manufacturer. Four volunteers  
who no longer smoked took lozenges from both sources  
on a schedule of two every 30 minutes and allowed  

them to dissolve without sucking. Subjects were meant  
to take 28 lozenges over seven hours but some stopped  
before this because of nausea. Blood samples for  
analysis of nicotine concentrations were taken 30  
minutes after the last dose. The mean plasma nicotine  
concentration achieved with supplies bought from a  
pharmacist was 14-6 μg/l after an average of 22  
lozenges taken over five and a half hours. The mean concentra-  
tion achieved with lozenges supplied by the factory was  
22-3 μg/l after an average of 17 lozenges over four  
hours. The plasma nicotine concentration increased by  
a mean of 4.6 μg/l (range 3.6-5.2) over 30 minutes in  
three subjects who took two lozenges supplied by the  
factory.

Comment

Stoppers led to substantial absorption of nicotine. The  
concentrations from lozenges bought locally were higher  
than those from clinical use of 2 mg nicotine chewing gum,  
whereas lozenges supplied by the factory gave concentrations similar to the lowest  
achieved from cigarette smoking and to those achieved  
from chewing 4 mg gum on an imposed schedule.  
Absorption from two lozenges was the same as  
depicted by the plasma concentration from  
that from one piece of 2 mg gum. The factory lozenges  
delivered more nicotine than those bought locally,  
confirming reports from patients and suggesting that  
the product may have a limited shelf life.

Our observations suggest that Stoppers have some  
therapeutic potential as a specific effect of nicotine in  
allievating withdrawal from tobacco and promoting  
smoking is now well established. The ease of taking  
the lozenges may make them suitable for  
dependent smokers who find chewing gum difficult  
or aversive. At the same time, there must be concern  
about the lack of information and guidance provided  
for the consumer and about the potential for abuse.  
The lozenges are not packaged in child proof  
containers, and the labelling does not mention nicotine,  
say why nicotine might be helpful, or point out any  
hazards of use.

1 United States Department of Health and Human Services. Nicotine addiction: a  

2 West RJ, Jarvis MJ, Russell MAH, Carruthers ME, Feyerabend C. Effect of nicotine  

3 Russell MAH, Sutton SR, Feyerabend C, Cole PV, Saloman Y. Nicotine  

4 McNab ME, Ebert RV, Mccosker K. Plasma nicotine levels produced by  

5 Benowitz NL, Jacob P, Savasampadi C. Determinants of nicotine intake while  

(Accepted 12 December 1988)