- All letters must be typed with double spacing and signed by all authors.
- No letter should be more than 400 words.
- For letters on scientific subjects we normally reserve our correspondence columns for those relating to issues discussed recently (within six weeks) in the *BMJ*.
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Could an outbreak of poliomyelitis occur in the UK?

SIR,—With the elimination of indigenous poliomyelitis from the United Kingdom any cases of paralytic poliomyelitis are most likely to be associated with vaccine or imported from endemic areas. The importation of wild type poliovirus strains in ostensibly immune populations is becoming a cause for concern.

The risk of infection comes from non-Sabin-like epidemic strains, which have caused outbreaks in Finland, Israel, and Oman: poliovirus type 3 in a population in Finland protected with inactivated poliomyelitis vaccine¹; poliovirus type 1 in Israel, where enhanced inactivated poliomyelitis vaccine superseded oral poliomyelitis vaccine²; and poliovirus type 1 in a population in Oman protected with oral poliomyelitis vaccine.³ The outbreak in Oman occurred against high uptake of vaccine and spread among vaccinated children.

In Glasgow we recently investigated a major outbreak of hepatitis A enteroviral infection in a community in a deprived housing scheme.⁴ We concluded that the spread of virus was possibly amplified by contamination of the environment with sewage from an infected household. Major outbreaks of enteroviral infection, including poliomyelitis, are associated with faecal contamination of the environment. We therefore measured levels of immunity to poliomyelitis in this community.

Serum from 24 of 47 subjects identified as having hepatitis A was tested for antibody to poliovirus at the National Institute for Biological Standards and Control. Antibody titres to type 1 and type 2 virus were above recommended levels; only 35% of the subjects, however, had this level of antibody titre to poliovirus type 3. Only two of the samples tested were from subjects aged over 30. Thus protection against poliovirus types 1 and 2 was most probably vaccine induced. It is unclear, however, why the antibody response to type 3 virus was poor and whether this community is at risk from an outbreak of poliovirus type 3 infection, especially as the aetiology in a recent case of fatal poliomyelitis in the United Kingdom remains unexplained.⁵

Concern about levels of immunity to polioviruses is not new. Two previous studies in the United Kingdom have shown decreasing antibody levels in children.^{6,7} The apparent ease with which hepatitis A virus spread in this deprived urban community, and the associated contamination of the environment with sewage, shows the urgent need for further seroepidemiological studies of immunity to polioviruses in the United Kingdom.

We thank Dr David Wood, senior scientist at the National Institute for Biological Standards and

Control, for his help in measuring the poliovirus neutralising antibodies and for his advice.

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Unstable asthma and theophylline

SIR,—The relation between plasma concentrations of theophylline, control of symptoms of asthma, and peak flow measurements was questioned in an interesting case history presented by Myra Stern, with the suggestion that a life threatening exacerbation occurred despite the maintenance of therapeutic plasma concentrations.¹

We agree that there are many variables in the home environment that affect home monitoring. The relative timing of administration of theophylline and assay, however, was not clear, making any analysis questionable.

After slow release theophylline was introduced the patient's asthma, in terms of peak expiratory flow, was stable for six weeks and a morning fall in peak flow was quickly corrected by dose adjustment. When theophylline was withdrawn and on another occasion when the patient suffered diarrhoea and vomiting, however, peak expiratory flow rates deteriorated rapidly. Diarrhoea and vomiting would have affected the absorption of all oral preparations the patient was taking, including theophylline and oral steroids. Interestingly, withdrawal of salbutamol had no deleterious effect on the control of peak expiratory flow rate.

The experience in this case is similar to that of Brenner *et al.*, who withdrew theophylline treatment in five young patients with severe asthma who were concurrently taking 400-800 mg of inhaled beclomethasone dipropionate and 10-30

mg of prednisolone daily.² Within 48 hours of stopping theophylline all five patients had an appreciable increase in symptom score and a considerable decrease in peak flow measurements and each required an increase in the steroid dosage. Three of the five patients had severe non-infective exacerbations, and the results were such that it was considered unethical to recruit further patients to the study.

Theophyllines have been used for many years to manage asthma, but only recently has research started to unravel the complexity of theophylline's activity. Recent work has shown that in addition to producing dose dependent bronchodilatation theophylline attenuates the late asthmatic response³ and modulates the inflammatory changes in the airways, principally through its action on T lymphocyte subsets.⁴ We are therefore not surprised that on stopping treatment the patients described by both Stern and Brenner *et al.* deteriorated much more than could be expected from the bronchodilator effects alone.

Contrary to Duncan M Geddes's opinion,¹ we consider that the only valid conclusions that can be drawn from this case study are that theophylline has an important role in the management of patients with unstable asthma and that its withdrawal in such patients may precipitate a severe exacerbation despite the continued administration of other appropriate treatments.

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Medically unexplained physical symptoms

SIR,—Though I agree that we should try to provide an explanation for persistent, mystifying physical symptoms, I am surprised that Samuel I Cohen can usually make a physical diagnosis in such cases.¹ Have I been missing the ischaemic jaws and contracting chest muscles of the anxious and unhappy, or is Cohen confusing the art with the science of non-disease?²

The interpretation of symptoms by patients and

doctors is at the core of the practice of medicine and is an important determinant of consultation behaviour and clinical care. Hannay made us familiar with the symptom iceberg,³ although only one in 40 symptoms becomes the subject of a medical consultation.^{4,5} The severity of symptoms is a poor predictor of a patient's likelihood to consult a general practitioner; studies of gastrointestinal symptoms have shown that concern about the implications of symptoms for serious or life threatening disease operates as a trigger to consultation.⁶ Thomas made the crucial observation that in up to 40% of patients seen in general practice it is impossible to make a firm physical diagnosis and that most of these patients recover completely and spontaneously; Thomas coined two telling phrases to describe this work—"the temporarily dependent patient"⁷ and "the therapeutic illusion."⁸

In general practice we are concerned to make early and accurate diagnoses of important illness and to avoid unnecessary investigation of minor and self limiting disorders. To answer the question "Well, if it's not serious then what is it?" we use what Dixon dubbed diagnostic black boxes, which apparently explain problems as long as you do not inquire into the mechanism too closely.⁹ Terms such as "a virus," "sprains," and "fibrositis" help to identify otherwise unidentifiable disorders and point the way to non-intervention.

The last piece of the jigsaw was provided by Thomas¹⁰; in patients in whom no definite diagnosis could be made a "positive consultation" (in which a definite diagnosis was nevertheless given) was associated with greater patient satisfaction and a higher recovery rate than an artificially "negative consultation."

Cohen's patients will undoubtedly benefit from his diagnostic certainty. A particularly elegant path between candour and paternalism was taken by the eminent specialist in the apocryphal quotation: "You want to know what's wrong. Well, it's not a condition to which we doctors can yet exactly put a name. But I want you to know this. You've come to the very best man in Britain for it."

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Passive smoking and otitis media with effusion

SIR,—The fact that G S Barr and A P Coatesworth found no association between parental smoking and otitis media with effusion shows that this difficult but important question is far from being completely answered.¹ Although in their case-control study they matched for age, sex, and social class, it is not clear over what period the two groups were collected. Children examined during the winter are more likely to have a middle ear

effusion, and this may have affected the results unless the two groups of children were collected simultaneously.

The balance of evidence from clinical studies points increasingly to an association between passive smoking and otitis media with effusion. When I compared a group of 115 children undergoing insertion of grommets for otitis media with effusion with a control group of 36 children from an orthoptic clinic I found a significant association between passive smoking and the need for grommets. Although this was not a case-control study, no significant differences were found between the two groups of children with regard to sex, age, or social class distribution.²

The Edinburgh study of 892 schoolchildren aged 7 also quoted in Barr and Coatesworth's paper shows an association between results of tympanometry suggestive of a middle ear effusion and passive smoking. The Edinburgh group concluded that in their study about one third of the cases of middle ear effusion were attributable to exposure to tobacco smoke.³ I agree with Barr and Coatesworth that tympanometry alone is not an absolute indicator of the presence of a middle ear effusion, but when efforts are made to exclude results showing high canal volumes (indicating a perforation of the tympanic membrane) and very low canal volumes (indicating a probe blocked with wax) a flat tympanogram is highly suggestive of a middle ear effusion.

Experimental studies are yielding further insights into a possible causative link between tobacco smoke and middle ear effusions. Constituents of tobacco smoke may lead to ciliary dysfunction,⁴ and an experimentally induced reduction in middle ear ciliary function may lead to the production of a middle ear effusion.⁵

Obviously, further research is warranted to confirm the probable relation between parental smoking and otitis media with effusion, and experimental studies are needed to explain the mechanism of any causative link.

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European health challenges

SIR,—The World Health Organisation's Health for All programme targets differences in health status between groups within countries. The aim is a 25% improvement in the health of the disadvantaged group (target 1). In Tony Smith's article on European health challenges differences between sexes are referred to only in terms of life expectancy.¹ Other evidence suggests that it is reasonable to target men as a particularly disadvantaged group. The Black report showed that the ratio of death rates for 15-64 year old men compared with women was 1.9 in 1971, but it did not have sex differences within its mandate.² This compared with a ratio of 2.5 for social class V compared with social class I. At the same time men's consultation patterns lead to fewer opportunities for doctors to be involved directly with the target population.³

Although morbidity and mortality among men

and women can be aptly summarised as "women suffer while men die," mortality statistics for men demand that men be treated as a disadvantaged group and targeted accordingly. This targeting must begin early while health behaviour is being established⁴ as at an early age boys are more likely than girls to expect to remain healthy.⁵

The disadvantages of the sexes are as serious as those associated with the different social classes and must be targeted likewise.

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Preventing AIDS now

SIR,—Robert J Biggar indicated that resistance to using condoms in many countries is due to reduced sexual pleasure, the belief that condoms encourage promiscuity, and religious or cultural beliefs. In a pilot study we found evidence suggesting that lack of knowledge regarding the effectiveness of condoms should be added to that list.

We performed a study in February 1991 at the Aga Khan Hospital, Nairobi, which draws patients mainly from social classes I, II, and III, the educated and wealthy classes. We interviewed 50 medical patients using a standard questionnaire. Twenty six were positive for HIV antibodies (four were unaware of their diagnosis) and 24 were negative for HIV antibodies or had not been tested. Only one patient refused to participate.

Knowledge regarding the risk of HIV transmission was good for some aspects and poor for others (table). Most worrying was the fact that only 55% believed condoms prevented the transmission of HIV infection. Furthermore, several patients also voiced concern about the risk of condoms splitting.

More effective health promotion strategies

Knowledge regarding HIV transmission in 49 Kenyan patients

Route of transmission	No of patients responding		
	Yes	No	Don't know
Blood transfusion	47	1	1
Sterile needle	6	39	4
Used needle	49		
Donating blood	19	27	3
Sharing a cup	8	36	5
Handshake	1	47	1
Kissing	19	26	4
Breast milk	24	7	18
Vaginal sexual intercourse	48		1
Oral sexual intercourse	20	14	15
Anal sexual intercourse	27	3	19
Masturbation of partner	15	25	9
Vaginal sexual intercourse with condom	18	27	4

regarding safe sex need to be developed and it is vital that this is accompanied by the ready availability of high quality condoms.

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