to the conclusion arrived at by Hodson⁸ after a detailed review of the international literature regarding the use and misuse of chlormethiazole in general—that is, also in geriatrics or obstetrics (pre-eclamptic toxæmia, eclampsia). No cases of dependence have been reported among geriatric or obstetric patients and those reported among alcoholics (and with sufficient detailed data available) referred as a rule to highly unstable personalities. It should therefore be stressed that the risk of chlormethiazole dependence is a real one when the drug is “misused” in (unstable) alcoholics but seems very low when it is used in the withdrawal phase of alcoholics in the correct way, and equally so when used among the non-“dependence-prone” segment of the population.

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- ¹ Bradley, J J, *British Medical Journal*, 1977, **2**, 774.
² Glatt, M M, *British Medical Journal*, 1977, **2**, 1088.
³ Horder, J M, *British Medical Journal*, 1978, **1**, 693.
⁴ Glatt, M M, *Alcoholism—A Social Disease*. London, English Universities Press, 1975.
⁵ Glatt, M M, George, H R, and Frisch, E P, *British Medical Journal*, 1965, **2**, 401.
⁶ Frisch, E P (ed), *Acta Psychiatrica Scandinavica*, 1966, **42**, suppl 192.
⁷ Glatt, M M, Lecture at the 22nd International Institute on Prevention and Treatment of Alcoholism, ICCA, Vigo, Spain, 1975.
⁸ Hodson, K, personal communication.

Breathing other people's smoke

SIR,—A recent study,¹ to which there was no reference in your leading article (12 August, p 453), sheds a little more light on the possible health risks of breathing other people's smoke. As part of a population study in three USA towns the contribution of familial factors, including smoking, to lung function and respiratory symptoms was investigated in 376 families with 816 children. Neither lung function nor respiratory symptoms of a non-smoking husband or wife were affected by a smoking spouse. Parental smoking appeared to have no effect on children's respiratory symptoms. The children themselves answered questions about cough and phlegm. In other studies which have shown an association between children's symptoms and parental smoking parents answered the questions for their offspring and this may have biased the results. Also in the USA population study¹ there was no convincing evidence that parental smoking adversely affected children's lung function. Out of five different measurements of lung function the only significant association was that daughters' maximum expiratory flow rates at 50% of forced vital capacity were lowest in families in which the mother smoked and the father was a non-smoker. The effect was small and could have been a chance finding. However, smoking parents compared with non-smokers had a significant reduction in forced expiratory volume in 1 s and the results for forced vital capacity and flow rates showed similar trends.

The only definite evidence that parental smoking may affect their children's respiratory system is that infants of smoking parents have more respiratory illness during the first year of life than infants of non-smoking parents,² but in the same population this association was absent in children aged 1-5 years. At present there is no firm evidence that these illnesses in children under one year have a serious and lasting effect as no excess of respiratory illness

or diminished lung function has been found in the older children of smoking parents.

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AREND BOUHUYS

Yale University Lung Research Center,
New Haven, Connecticut

- ¹ Schilling, R S F, et al, *American Journal of Epidemiology*, 1977, **106**, 274.
² Colley, J R T, Holland, W W, and Corkhill, R T, *Lancet*, 1974, **2**, 1031.

SIR,—In your leading article on this subject (12 August, p 453) you write that “small amounts of nicotine inhaled by non-smokers are unlikely to have any medical importance.” In a recent publication in *Allergie et Immunologie*¹ I describe an atopic dermatitis affecting five members of my family who are clinically hypersensitive to the nicotine alkaloid of *Nicotiana tabacum*. We have a specific degranulation of the basophils (DIB test of Benveniste²) with the hapten nicotine bound to human serum albumin and the passive cutaneous anaphylaxis test is strongly positive in the rabbit. A single puff of tobacco smoke can induce an anaphylactic reaction of the skin—“atopic dermatitis” or “asthma of the skin” (Tuft).

We are at present carrying out investigations with Professor A L De Weck, of the Institute for Clinical Immunology in Berne, the results of which will be published shortly.

BERNARD J-L SUDAN

Basle,
Switzerland

- ¹ Sudan, B J-L, *Allergie et Immunologie*, 1978, **10**, 36.
² Benveniste, J, *Annals médicales de Nancy*, 1977, **16**, 49.

SIR,—Your leading article (12 August, p 453) explained that two cases of lung cancer per 100 000 per year are caused by the benz(a)-pyrene in environmental tobacco smoke, that environmental tobacco smoke may endanger persons with coronary heart disease, and that children of smokers have more upper respiratory tract infections than children of non-smokers. Yet you conclude that breathing other people's tobacco smoke does not pose “serious risks to health.” I feel certain that those two persons with lung cancer caused by environmental tobacco smoke, those many persons whose attack of angina pectoris was exacerbated by environmental tobacco smoke, those children of smokers with upper respiratory infections, and those thousands of persons with either pre-existing respiratory ailments or allergies to tobacco smoke would believe that tobacco smoke *did* pose a serious risk to their health. However, the issue is not whether tobacco smoke poses “serious” risk to non-smokers; it is whether smokers have a right to expose non-smokers to *any* risk. Tobacco smoke, unlike emissions from automobiles or factories, is not the product of a socially necessary activity.

Finally, you state that since environmental tobacco smoke does not pose serious risks to health, non-smokers' assertion of “the right to breathe smoke-free air must be based on aesthetic considerations.” Legal considerations are totally neglected. At English and American common law a battery is the “intentional, unpermitted, offensive touching of the person of another,” regardless of whether such touching causes harm to the health of

the other. The legal harm is the offensiveness of the touching (kissing an unwilling party may be a battery). The touching may be with an instrument (a rock, smoke). Your readers have social and legal as well as medical grounds to insist on smoke-free air.,

BETTY BRODY
Attorney at Law

Swampscott, Massachusetts

* * * This correspondence is now closed.—ED,
BMJ.

Uniform style for biomedical journals

SIR,—So far your published correspondence on this topic has not included the views of those likely to benefit the most—namely, biomedical writers and their long-suffering secretary/typists. Perhaps they are afraid to put their names on record for reasons of modesty? Not being so inhibited, we are presuming to write on behalf of the myriads who have spent countless hours of work in adapting and readapting the style of references. Such a morass of minute and conflicting requirements is so ridiculous as to be laughable and so expensive of time and money as to be a reproach to our joint sense of responsibility.

The International Steering Committee of Medical Editors (20 May, p 1334) deserves not merely thanks and appreciation but, wherever it might be effective, wholesale support. For instance, those of us who submit manuscripts could invariably adopt the recommendations (including any subsequent refinements or modifications).

Perhaps the day may come when you, Sir, and your fellow editors will find further modification unnecessary. There will be little lost but much would be gained.

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J LOVEDAY
M Y MURPHY

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Harrow, Middx

Misdiagnosis of amoebiasis

SIR,—It was with some concern that I read the letter from Dr T H Foley and his colleagues (5 August, p 428) and your leading article on the same subject (p 379), both of which appeared to undermine public confidence in the availability of reference services for the diagnosis of amoebiasis.

At the invitation of Sir James Howie, at that time Director of the Public Health Laboratory Service, a reference service for the diagnosis of this potentially lethal but curable disease was instituted at this hospital in October 1969. The service has remained freely available to every hospital and microbiology laboratory in the United Kingdom and is in fact used regularly by the very hospital from which Dr Foley writes. Of the many thousands of requests for investigations relating to the diagnosis of amoebiasis since the service was begun, none has ever been declined.

The service has been listed annually, together with other diagnostic services, in the *PHLS Year Book and Directory*, copies of which are available to every hospital laboratory throughout the country. Judging by the ever-increasing (though welcome) demands made

on the service from all parts of Britain and many countries overseas, however, it is evident that its existence is well known, at least to the great majority of consultant microbiologists in the United Kingdom.

Since Dr Foley's letter and your leading article regrettably appear to give an entirely false impression of the available facilities I should like to make it clear that the service provided at Guy's remains fully active, is publicised through the proper channels, and is freely available to all who need to use it.

A L JEANES

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Melatonin as a marker for pineal tumours

SIR,—In our short report (29 July, p 328) my colleagues and I recorded marked elevations of circulating melatonin levels showing a diurnal variation in a patient with a proved pineal tumour. It is difficult to explain these findings in the context of current ideas outlined in Dr E Tapp's letter (26 August, p 635) but we wholeheartedly agree that "the mechanisms controlling melatonin secretion . . . are . . . incompletely understood"—and merely offer tentative explanations. As far as we could judge and as stated in the report, normal pineal tissue could not be found at necropsy and, although it is possible that some may have been inadvertently included in the tissue enzyme assays, this seems improbable. Perhaps identification of the precise origin of the circulating melatonin will have to await the development of immunofluorescent antibody techniques for melatonin in tissue slices.

It is interesting that Dr Josephine Arendt (p 635) has also found a patient with a pineal tumour and high melatonin levels, though it is disappointing that no clinical details are available. Since the initial report we have published details of an additional five patients,¹ all of whom resembled the first in having mid-day serum melatonin levels at least five times normal. As we have suggested, further study of the effects of other diseases and of radiotherapy is necessary before the full value of our observations can be determined, but at the present time we believe our results to be consistent and promising.

S G BARBER

General Hospital,
Birmingham

¹ Barber, S G, *et al*, *Lancet*, 1978, **2**, 372.

Whooping cough

SIR,—I am grateful for Professor G T Stewart's observations (9 September, p 768) on the Keyworth whooping-cough study (19 August, p 577) and I am pleased to answer his and Professor J A Davis's points.

The population studied is remarkably isolated, geographically and socially, for a semirural area. My partners and I can recall only one case of whooping cough in the years 1974-5 and I would suggest that the 1974 cohort was not exposed to whooping cough then. The 1977 cohort, which shows the lowest attack rate, had, I believe, the lowest rate of social interaction with affected individuals. My observation of the spread of the disease in the first two months of the

Nasopharyngeal swabs

Age (years)		0-1	1	2	3	4	5	6	7	8	9	10	11	12	Adult	Total	
<i>Bordetella pertussis</i> isolated	Immunised	0	0	0	0	0	5	2	0	1	1	0	0	0	0	9	23
	Non-immunised	1	5	3	3	0	0	1	0	0	0	0	0	0	1	14	
<i>Bordetella pertussis</i> not isolated	Immunised	0	0	1	4	3	4	2	3	1	1	2	2	1	0	24	34
	Non-immunised	1	3	3	3	0	0	0	0	0	0	0	0	0	0	10	

outbreak demonstrated acquisition of the disease only from recognised cases—between siblings, neighbouring children, relatives, etc—and a closer look at the affected members of the 1977 cohort shows a consistent picture. There were 10 families in the community with a child born during 1977 in which there was also an older child who developed whooping cough. Eight of these infants developed the disease, none of whom had had any pertussis immunisation. A further case was in an only child who was in contact with a child next door with whooping cough. Two of the 10 infants exposed did not develop the disease. One had had two doses of DPT and the other was given prophylactic erythromycin (this being the only instance of antibiotic prophylaxis in the study).

The attack rate in immunised siblings under 5 was 20%, in marked contrast with the non-immunised, in whom it ranged from 50% in those born in 1973 to 88% in those born in 1976. Positive swabs were obtained more frequently from the non-immunised than from the immunised (see table above).

The group of 20 serious cases, as defined by the presence of cyanotic attacks in children under 5 years old, breaks down as follows: seven aged 1 year, eight aged 3 years, and five aged 4 years. Three lived in three-bedroomed terraced council properties and 17 in owner-occupied detached or semidetached houses.

DOUGLAS JENKINSON

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Arenaviruses in perspective

SIR,—Your leading article (4 March, p 529) brings the arenaviruses into perspective and at the same time raises some interesting epidemiological and diagnostic features of the disease syndrome. As you state, all these viruses cause remarkably similar symptoms. In addition to the arenaviruses and Marburg and Ebola viruses mentioned by you, haemorrhagic fever (HF) syndrome may also be due to leptospirosis, bacterial septicaemia, and viruses like yellow fever, dengue, chikungunya (mosquito-borne), Kyasanur forest disease, Omsk haemorrhagic fever, and CHF-Congo (tick-borne).¹ Because the disease is not characterised in the early phase a major problem is of differential diagnosis, particularly as the areas involved are far from free of other common pathogens.

Considering the overall epidemiological, clinical, and immunopathological features of dengue haemorrhagic fever (DHF) in South-east Asia I proposed a dual aetiology—interaction of virus and parasite infecting a human host around the same time.² It would not be too far-fetched to suggest a similar phenomenon for the serious manifestations resulting from arenaviruses and other viruses causing HF. This would explain the differences noted in clinical features—for example, gastrointestinal haemorrhages in one region, a

renal syndrome in another—as being due to the prevalence of not only a certain type of virus but also a particular type of parasite in a given region. Within a particular region the wide spectrum of clinical manifestations caused by infection with a virus may be dependent upon whether there are simultaneous infections, as also on the type and the stages of these infections.

According to this concept the ultimate outcome would be dependent on the host's immune response to such infections. For the pathogenesis two types of hypersensitive reaction are envisaged: (1) deposition of antigen-antibody complexes (both viral and parasitic) leading to complement activation and local inflammation; and (2) reagin- or IgE-mediated release of histamine from the sensitised cells leading to increased vascular permeability and vascular collapse.

Heavy parasitisation being common in the involved regions the IgE levels would be expected to be high to begin with. However, we could demonstrate significantly higher values for IgE in the sera of DHF patients from Bangkok as compared with controls.³ These were observed from the early phase of the illness, around the third day. It is suggested that an early test for IgE may help in predicting a grave prognosis among those patients who show significantly higher levels of IgE than the levels normally encountered in a particular region.

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¹ WHO Weekly Epidemiological Record, 1976, **51**, 325.

² Pavri, M, *Indian Journal of Medical Research*, 1976, **64**, 713.

³ Pavri, K M, *et al*, *Indian Journal of Medical Research*, 1977, **66**, 537.

Underdiagnosis of childhood asthma

SIR,—How often, in general practice, do we wait for our hospital colleagues to highlight a theme which is really our concern? Dr A N P Speight's revelations (29 July, p 331) about the underdiagnosis of childhood asthma have spurred us to report the current status of this condition in our own practice, which now operates a comprehensive morbidity register. We hope to publish a fuller report at a later date, but in the meanwhile your readers will be interested to know that asthma is the "market leader" in our chronic morbidity list (No 2 is hypertension). In fact 2.4% of the practice list of 7451 patients are registered as asthmatics and 36% of these asthmatics are children in the 0 to 14 age range. Moreover, random sampling suggests that 90% of these children were diagnosed as such before the age of 6 years.

Dr Speight correctly highlights some of the negative factors blocking the diagnosis. We would like to emphasise some positive factors which we believe have led us to make the diagnosis perhaps more often than other