

results suggest an accumulative effect," but they continued: "The mean oestrogen levels were within the premenopausal range after six successive implants, even at the time of recurrence of symptoms."⁷

We acknowledge that Mr Studd and his team have reported the return of symptoms in the presence of premenopausal plasma oestradiol concentrations. None of these papers emphasised, however, that implants in postmenopausal women can result in supraphysiological oestradiol concentrations and mentioned the failure of further implantation to control symptoms adequately with decreasing durations of benefit.

Mr Studd and coworkers accuse our paper of having "an obvious sampling bias." This we cannot understand because owing to the limitations of a short report we presented few demographic or historical medical data on our patients. This is acknowledged by Mr Studd and coworkers, who later in their letter request specific information on our study population. Three of our 12 patients had undergone bilateral oophorectomy under age 35, two presented with depression, and six were perimenopausal (flushes and sweats, but still menstruating, albeit erratically). None had a psychiatric history. Initially, all of our patients derived good relief of symptoms from six monthly insertion of implants. Therefore we disagree with Mr Studd and coworkers that "tachyphylaxis will be found mostly in women with psychiatric pathology."

We stated that the optimal management of implant tachyphylaxis is not known, and we suggested withholding all forms of oestrogen until the plasma oestradiol concentrations had returned to approximately 200 pmol/l. This concentration is considered "inadequate for most patients" by Mr Studd and coworkers, yet vasomotor symptoms have been shown to be relieved at such concentrations and the psychological state and vaginal cytology in postmenopausal women to be improved.^{8*} Most oral and transdermal oestrogen preparations prescribed in the United Kingdom give plasma oestradiol values around this concentration. From a prospective, randomised study we now have data that bone mass in the spine and hip will be conserved at such concentrations compared with that in an untreated reference group (unpublished data).

We agree that this level is likely to be inadequate in premenopausal women (who are exposed to higher endogenous oestradiol concentrations) treated by implants for premenstrual syndrome; and, clearly, further oestrogens will be required in postmenopausal women if severe symptoms such as depression recur. Each case will have to be judged on its own merits. Because our study group derived benefit for shorter and shorter intervals with further implantation and also developed symptoms of oestrogen overdosage we see no alternative to withholding oestrogens for prolonged periods in most women who develop tachyphylaxis. We, too, question the ethics of using these patients to investigate the pharmacokinetics of oestradiol implants and have therefore studied a separate group of women to determine the duration of endometrial stimulation after implantation.⁹ (Patients in this additional study were those who needed to discontinue treatment for valid medical reasons, such as breast cancer, or who had expressed a desire to stop treatment.)

We suspect that the problems that we described are not uncommon. Most of the delegates attending the menopause session at the recent British congress of obstetrics and gynaecology (chaired by Mr Studd) signified by a show of hands at the meeting that they had encountered "tachyphylaxis." Oestrogen replacement therapy is indeed an important form of preventive medicine, and we are not suggesting that implants should be abandoned. It seems sensible, however, to try to reduce the occurrence of their side effects, especially if these

are largely iatrogenic. Regular monitoring of oestradiol concentrations when appropriate and resisting the temptation to insert further implants when these concentrations are excessive will help to avoid tachyphylaxis, which is intensely distressing to the patient and presents a management dilemma to the clinician.

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

Housing and health

SIR,—The article by Dr Stella Lowry makes the important point that although moderately cold and damp housing is unpleasant, it is not certain that it increases respiratory symptoms.¹ It could be added that one report suggests that fewer respiratory symptoms are found among children in homes without rather than with central heating.² There are indications, however, that central heating has reduced more severe respiratory disease.³ Provision of central heating increased dramatically, from 13% to 66% of homes, in England and Wales between 1964 and 1984. This was accompanied by a 69% decline in excess winter mortality from respiratory illness (as a fraction of summer mortality, with allowance for variations in coldness of winters and for a constant age group of 70-74 years), and this could only partially be attributed to a decline in epidemics of influenza. By contrast, the more numerous excess winter deaths from coronary thrombosis and cerebrovascular accident have not fallen significantly with the improvement in home heating and may be more related to changes in blood composition caused by episodic outdoor exposures to cold. The evidence therefore does not make it possible to draw up a full balance sheet of the effects of warming houses but does seem to justify promoting measures for avoiding cold discomfort both indoors and outdoors, on grounds of health as well as comfort, at least among elderly people.

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- 1 Lowry S. Temperature and humidity. *Br Med J* 1989;299:1326-8. (25 November.)
- 2 Yarnell JWG, St Leger AS. Housing conditions, respiratory illness and lung function in children in south Wales. *British Journal of Preventive and Social Medicine* 1977;31:183-8.
- 3 Keatinge WR, Coleshaw SRK, Holmes J. Changes in seasonal mortality with improvement in home heating in England and Wales from 1964 to 1984. *Int J Biometeorol* 1989;33:71-6.

SIR,—Dr Stella Lowry's article on temperature and humidity highlighted the deleterious effects of these weather variables on health.¹ Most readers are familiar with the excess winter mortality seen in Britain primarily among the elderly and its attribution to respiratory and cardiovascular disease, but how many are aware that the first part of the respiratory tract to come into contact with the seasonal changes in ambient atmospheric conditions shows morbidity beyond winter coryza?

A study of 686 patients hospitalised over a two year period in the greater Glasgow area for idiopathic spontaneous epistaxis found a correlation of -0.81 between admissions and ambient temperature ($p < 0.01$). There was no correlation with atmospheric pressure, hours of sunshine, or relative humidity.²

Reporting bias of patients in this study is unlikely to be high as only patients who required hospitalisation for periods of 24 hours or longer as opposed to outpatients were considered, and there is no reason to believe that there is in Glasgow a large group of people with severe epistaxis who fail to seek medical attention.

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- 1 Lowry S. Temperature and humidity. *Br Med J* 1989;299:1326-8. (25 November.)
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SIR,—The results of my three year study looking at housing and health in east London support Dr Stella Lowry's conclusion that damp, cold housing may affect people's health.¹

I conducted two surveys taking a range of subjective and objective measurements of aspects of housing and health. Objective measurements included peak flow rate as an indicator of respiratory health; readings from a whirling hygrometer as spot measures of temperature and humidity; and spore counts. Thermohydrographic measurements were used to obtain weekly averages of temperature and humidity in the second survey.

Table I shows the results of the first survey. Similar associations were found in the second survey of 60 households, including significant relations between reported damp and cold and average weekly measurements of temperature and humidity. This suggests that self reported measures may be useful and valid.

TABLE I—Relative humidity, temperature, spore counts, and peak flow rate in households reporting and not reporting cold, dampness, mould, and respiratory illness

	Measurement	p Value
Relative humidity (%) (n=204):		
Dampness	63.8	
Not damp	55.9	<0.0001
Temperature (°C) (n=204):		
Cold	12.6	
Not cold	15.4	<0.0025
Spore counts/m ³ (n=204):		
Mould	25 596	
Not mouldy	10 274	<0.0087
Peak flow (%) (n=262):		
Chest problems*	80.0	
No chest problems	86.1	<0.0428
Peak flow (%) (n=249):		
Hidden asthma†	78.5	
No hidden asthma	90.8	<0.0001

*Bronchitis, chesty colds, or asthma.

†Coughing during day or night, wheezing, breathlessness, or blocked up nose.

Dr Lowry mentioned the role of occupiers in the prevalence of dampness in the home. In my second survey I compared households with and without central heating. There were no significant differences between the two groups regarding lifestyle factors that might influence humidity—for example, household size, drying of clothes, wash-