whose ostensible aim is to promote "responsibility."

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Housing, health, and illness

SIR,—The data from Mr H F Thomas and Dr J W G Yarnell (29 July, p 358) suggest that medical intervention in housing in Oxford (8 July, p 100) is comparatively ineffective and, presumably, inefficient. Before raising the question of the extent to which housing should be medicalised, I would like to comment on the possible meanings of the data presented. In Oxford, medical points account for about 5% of the points level of the qualifying threshold for housing from the waiting list, whereas they account for over half the level of the qualifying threshold in some of the Welsh authorities cited. It is possible that this is due not to any lesser respect for medical opinion in Oxford but to the fact that the city of Oxford is politically evenly balanced. Neither party predominates and, in my opinion, because of this a system has evolved in which the housing need of applicants is considered principally on falsifiable criteria—for example, the size of the rooms they occupy and the number and age of their children. There is little scope for discretionary assessment of need or "merit" for housing by individual councillors, housing managers, and medical advisers. The time which will elapse before an applicant is rehoused is therefore determined mainly by criteria which have been debated and agreed by both political parties and published for all applicants to consult. Could it be that those authorities in which the power of medical discretion was great were one-party councils in which there is greater discretion of all sorts?

The cases taken to committee can be regarded as those in which medical points accounted for 100% of the number required to qualify for housing. The opportunity to bypass the points system completely is useful not only because some cases occur which required immediate action, but also because it gives me a valuable chance each month to discuss cases, policies, and principles with councillors.

The points raised by Dr F J G Lishman (5 August, p 431) are valid, but I still do not think that I could grade "urgency" with confidence after a single home visit. The family knowledge that their chance of rehousing was a function of the degree of urgency they presented to a visitor means that such knowledge could never be objective. In my opinion such visits introduce other personal relationships and therefore other biases into the assessment.

That my observations are inferences and limited, as Drs D S Pickup and S Moolerjee suggest (5 August, p 431), is correct, but I hope they are valid, for all inferences are limited. The number of mental health cases dealt with has decreased over the years as the social services department now has the right to bring cases to committee, a progressive move by the city of Oxford which has demedicalised one aspect of housing need. It is interesting that the proportions of cases helped in Liverpool and Oxford are so similar, perhaps because of the equal political balance in both cities.

Would I not like more power? Should I not be using all the Aesculapian authority I can command to advance those cases referred to me as vigorously as I can? I think my answer to both questions is no. One reason is that even the system of self-referral employed in Oxford does not ensure that all the applicants with medical problems are known to me. No individual can identify all the possible need in his catchment area, but rather than devote more energy to the promotion of those cases known to me, which may be strong cases or may be cases with strong advocates, I am trying to identify more of those in need. Some applicants have no advocates at all. There are families in every city centre who are neither registered with a general practitioner nor literate. Also, I believe further medicalisation of housing is inappropriate in the city of Oxford. Housing need is not an illness; it is a human predicament which can be assessed as well by elected representatives and housing managers as by someone who has had a medical training. There is a continuing need for medical influence on cases and policy, but the form of that influence should be a function not only of possible relationships between housing conditions and the development of illness, but of the particular social and political context in which a community physician is working.

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Prevalence of anorexia nervosa

SIR,—In your Parliamentary news (15 July, p 213) you quote statistics concerning the prevalence and recorded mortality of anorexia nervosa. You report that "the prevalence of anorexia nervosa is about 1 per 100 of 16 to 18-year-old girls" and list the recorded deaths as ranging between 21 and 26 per annum in the past five years. These figures were produced by the Health Minister, Mr Roland Moyle, in reply to questions following a statement made in the House of Commons in which concern was expressed at the apparent increased incidence of the condition in recent years and the inadequacy of treatment facilities. The Health Minister apparently advanced the mortality statistics as evidence that there was no such increase.

I think it likely that the stated prevalence statistics derive from our own study,¹ which in fact revealed that the prevalence among 16 to 18-year-old schoolgirls was 1 per 200. It was 1 per 100 for those in the independent sector of education and 1 per 330 in the comprehensive sector.

The recorded mortality figures are misleading. Firstly, deaths from anorexia nervosa usually occur after five or more years of illness. A recent increase in incidence would therefore not yet be reflected in an increased mortality. Secondly, deaths in people with anorexia nervosa are not often recorded as caused by anorexia nervosa. Such people may have successfully concealed their condition to the last. Even if it is evident, then death may still be recorded as occurring only from suicide (the common cause of death in people with anorexia nervosa) or from one of the physical complications of the condition.^{2,3} Two patients with severe anorexia nervosa attending our clinic have died within the past few weeks. In the first death was recorded as

due to carbon monoxide poisoning. In the second it is likely that a similar cause will be recorded.

In the past few years this unit has received between 12 and 20 new requests per week to see patients thought to have anorexia nervosa. In the event we can take on one new such patient and her or his family per week, bearing in mind the intense and protracted treatment programme that usually stems from such consultations. Many people now hold the view that anorexia nervosa is becoming more common.

About four years ago two of us approached the Department of Health with a proposal that more extensive specialised services were needed for the treatment and management of this condition, but to no effect. People with anorexia nervosa, reluctant patients at the best of times, often continue, like alcoholics, to ricochet around the general medical and psychiatric services as and when they experience crises in their condition. In my view the problem has become much greater in the last few years because of the increased incidence, and the Health Minister would be well advised to reconsider the matter.

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¹ Crisp, A H, Palmer, R L, and Kalucy, R S, *British Journal of Psychiatry*, 1976, **128**, 549.

² Crisp, A H, *Proceedings of the Royal Society of Medicine*, 1977, **70**, 464.

³ Crisp, A H, *Proceedings of the Royal Society of Medicine*, 1977, **70**, 686.

Disodium cromoglycate ointment in atopic eczema

SIR,—Disodium cromoglycate (DSCG) is an effective inhibitor of IgE-mediated hypersensitivity reactions and has been used with success in patients with respiratory hypersensitivity of the type III as well as type I category.¹ More recently the use of topical 10% DSCG ointment in atopic eczema has been proposed, and a randomised double-blind trial of this treatment in 21 children with atopic eczema revealed a significant suppression of pruritus in patients receiving the active preparation, which was followed by improvement of the eczema.² However, the children studied in this trial had changes mainly confined to the skin of the limbs, with little or no involvement of the face and trunk. The response of more severe forms of atopic eczema of the type routinely referred to a dermatological clinic was therefore of interest.

Eleven patients (eight male) ranging in age from 13 to 38 (mean 20) years were studied. Only inpatients with eczema of moderate or severe degree who had given informed consent were admitted to the study. Six of the patients had associated asthma and had benefited from DSCG inhalation for this complaint. Each patient was allocated two outwardly identical tubes of ointment (white soft paraffin with 10% w/w DSCG or white soft paraffin alone), one marked for the left side and one for the right side, to be applied to affected areas twice daily for four weeks. The application was randomised so that some patients treated their left and some their right sides with the active ointment. The contents of the tubes were unknown to either the patient or the clinician. During and after a run-in period of five days, during which all previous topical therapy was stopped, the patient kept a diary of itching on both sides of the body and the clinical responses of the two sides were assessed weekly by the clinician and by the patient.

Six patients completed the study. In the remaining five the trial had to be abandoned because of acute flare-up of eczema on both sides (4) or severe itching on both sides (1). In none of the remaining six was there any significant difference in the itching or severity of eczema between the two sides. Only one of these patients showed an overall clinical improvement during the period of the trial.

Although numbers were small in this study the results suggest that at any rate in the formulation we used, DSCG is unlikely to be of therapeutic value in severe atopic eczema. The possibility remains, however, that topical DSCG may be of value in milder forms of eczema and possibly in reducing the incidence of relapses.

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¹ Pepys, J, *et al*, *Lancet*, 1968, 2, 134.

² Haider, S A, *British Medical Journal*, 1977, 1, 1570.

Physiological aspects of the menopausal hot flush

SIR,—As part of a study of the circulation in women after the menopause we also have been examining the vascular accompaniments of the "flush." Our findings, however, conflict with the conclusion of Mr D W Sturdee and others (8 July, p 79) that "the onset of the hot flush is associated with a sudden and transient increase in sympathetic drive."

Our studies, which included measurement of blood flow in the different segments of the limbs by venous occlusion plethysmography, have shown widespread circulatory change involving both capacitance and resistance vessels, the increased flow in the distal parts of the extremities suggesting that there is a release of sympathetic tone with the "flush" but not an increase. Premonitory symptoms of the "flush" may also be associated with alterations in blood flow. The timing of the vascular effects and their precipitation by stress would suggest a central origin for the response.

For rational therapy it is obviously important to establish the nature of the underlying physiological disturbance. In the light of our findings, however, drugs which influence peripheral vascular responses as the result of a central action would seem more likely to be effective alternatives to oestrogen for patients in whom the latter are unacceptable or contraindicated. We are therefore currently assessing the influence of therapy for menopausal symptoms on vascular reactivity using methods developed in previous assessment of drug effects in man.¹⁻³

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¹ Clarke, R S J, Ginsburg, J, and Hellon, R F, *Journal of Physiology*, 1958, 140, 318.

² Ginsburg, J, and Cobbold, A F, in *Adrenergic Mechanisms*, ed J R Vane, G E W Wolstenholme, and M O'Connor, p 173. Ciba Foundation Symposium. London, Churchill, 1960.

³ Beaconsfield, P, Ginsburg, J, and Rainsbury, R, *New England Journal of Medicine*, 1972, 287, 209.

SIR,—I was very interested to read the paper by Dr D W Sturdee and others (8 July, p 79) describing the progress being made in the study of the physiology of menopausal flushing. The work performed does appear

to verify the widely held clinical impression that menopausal hot flushes do have a basis of disturbed physiology at the vascular level which differs from heat-produced changes. Their use of heat study to provoke the flushes may, however, complicate the final analysis in that heat-induced vascular changes have themselves a well-researched physiology and flushes may be provoked in many other ways.

The hope expressed by the authors at the end of their paper that "improved understanding of the changes associated with the hot flush will lead to the development of a more specific and safer alternative to oestrogen" has, to a large extent, been pre-empted by our research into the effects of clonidine (Catapres; Dixarit),¹⁻³ following the work of Zaimis and Hannington,⁴ who showed that clonidine exerts a stabilising effect on small blood vessels making them largely insensitive to vasodilator and constrictor effects. Following the publication of our work this safe drug has been widely used with a good degree of success in menopausal flushing both in Britain and abroad.

One noteworthy point not published in the original report but which may be of value with other physiological studies was the observation of an interesting variation in the placebo response. This has to some extent been echoed in trials with oestrogen compounds both before and since our paper.⁵⁻⁷ Our results, however, indicated that the placebo response obtained varied markedly with the duration of the symptoms, being greater in patients who had suffered for more than one year, and much less in those who had suffered for less than one year. This may itself indicate the gradual establishment at the menopause of a microvascular physiological process whose response to any therapeutic stimuli affecting sympathetic or other humoral factors increases with time. It is to be hoped that further studies will be able to take all these factors into consideration and thereby help to produce a clearer understanding of a difficult area. In this respect clonidine itself may have a useful part to play as a physiological investigational tool.

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¹ Clayden, J R, *Lancet*, 1972, 2, 1361.

² Clayden, J R, Symposium on "The Migraine Headache and Dixarit," Churchill College Cambridge, 1972.

³ Clayden, J R, *et al*, *British Medical Journal*, 1974, 1, 409.

⁴ Zaimis, E, and Hannington, E, *Lancet*, 1969, 2, 298.

⁵ Pratt, J P, *Journal of the American Medical Association*, 1937, 109, 1875.

⁶ Coope, J, Thompson, J M, and Poller, L, *British Medical Journal*, 1975, 4, 139.

⁷ Campbell, S, and Whitehead, M I, *British Medical Journal*, 1977, 4, 31.

Treatment of hypertensive emergencies with oral labetalol

SIR,—With reference to the short report by Dr R R Ghose and others (8 July, p 96), I am not clear as to what relevance the title has to the data given in the article.

The title describes hypertensive emergencies. The article describes 11 consecutive patients with essential hypertension, one of whom had hypertensive encephalopathy. This is a hypertensive emergency, as would be acute renal failure or acute left ventricular failure. A diastolic pressure exceeding 130

mm Hg is not in itself a hypertensive emergency.

There is no doubt that in a hypertensive emergency the blood pressure should be controlled within minutes. Waiting two hours for a satisfactory response is, I believe, unacceptable in an emergency. The majority of beta-blockers are capable, when given orally, of producing a reduction of blood pressure within two hours.

In conclusion, the data given in this article did not support the title's suggestion that hypertensive emergencies may be treated with oral labetalol. I should welcome the authors' comments.

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* * * We sent a copy of this letter to Dr Ghose and his colleagues, whose reply is printed below.—Ed, *BMJ*.

SIR,—Our title (8 July, p 96) was intended to describe a therapeutic regimen for patients with severe hypertension who presented on emergency medical intake. We apologise if we misled Dr Good. Only two of these patients developed hypertensive crisis: one with visual blurring from papilloedema and the other from encephalopathy. Treatment caused rapid regression of symptoms in both cases. The latter, a 50-year-old previously fit man, was admitted as an emergency with severe headache and unexplained somnolence of sudden onset. Initial diastolic pressure was 110 mm Hg and the optic fundus showed grade II retinopathy. Central nervous system investigation showed no evidence of cerebral space-occupying lesion or infection. The blood pressure fluctuated and gradually rose to severe levels with a simultaneous decline in conscious level. Oral labetalol swiftly brought down the pressure (see figure) and produced prompt and permanent clearing of the sensorium with associated relief of headache.

Effect of oral labetalol on blood pressure and pulse rate in patient with hypertensive encephalopathy

Severe hypertension invariably requires antihypertensive therapy. The intaking clinician, unaware of the duration of preceding hypertension, needs to reduce arterial pressure below an ill-defined critical level at which vascular damage ensues. Whether this should be attempted over minutes with infusions of labetalol, diazoxide, sodium nitroprusside, clonidine, etc, or over hours with oral labetalol remains debatable. It is difficult to state categorically that the patient with hypertensive encephalopathy would have responded