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