prodromal and headache stages.[2] In each case the cortex perfusion rates were measured simultaneously in both hemispheres during the attack and later when the patients were fully recovered. I found a reduction in perfusion rate of 22·5% during the prodromal phase ($P>0·005$), but only a small, though significant, increase of 8·3% ($P>0·02$) in the headache stage. This small increase is quite compatible with a reactive hyperaemia following vasoconstriction in the prodromal stage, but is hardly sufficient to cause symptoms such as headache. Drs. Skinhøj and Paulson found a 47% increase in flow in the headache stage compared with their normal values, but this figure should be treated with some caution, since the resting flow in their patient is not known and this, together with the error associated with the method, means that no certain conclusions can be drawn from a single observation in one patient.—I am, etc.,

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Depressive Changes after Fluphenazine Treatment

SIR,—The development of long-acting phenothiazines is an important technical advance in psychopharmacology. As Drs. R. de Alarcon and M. W. P. Carney point out in the introduction to their report on depressive mood changes following the use of slow-release intramuscular fluphenazine (6 September, p. 564), it is becoming increasingly popular as the method of choice in maintaining schizophrenics in the community. Severe depressive reactions as a result of the drug would be a setback in this therapeutic progress, but, despite their detailed evaluation, the case is not very convincing. Fluphenazine is not a new drug. It has been available, and widely used, in oral form for ten years. If it were as potentially depressant as they suggest it is surprising that this has not been noted before. The long-acting injectable drug is still fluphenazine, and there is no evidence of pharmacological alteration by this technical change in the mode of administration.[1]

The phenothiazines as a group, considering their very widespread use in psychiatry, have not been reliably associated with genuine depressive mood changes. The anergic consequences of phenothiazine therapy in some schizophrenics have been described as depressive reactions, but they differ qualitatively from the depression of a depressive psychosis or the genuine depressive reactions that can occur with reserpine. If, as suggested, long-acting forms of phenothiazines behave differently in this respect the pharmacological explanation is not obvious.

Unless there is some acceptable explanation why long-acting injectable forms should be different from oral forms, it might be better to rely on the more obvious fact that mood disturbance is a common feature of schizophrenia and suicide is a not uncommon outcome of this disease. It is widely recognized that suicide is a particular danger during the treatment phase in depressive psychoses, owing to the alleviation of retardation in advance of the depressive thoughts. We do not conclude from this that electric convulsion therapy or antidepressants are depressant because of the suicidal incidence following their use. The schizophrenic under treatment may well be at risk owing to clinical changes such as returning insight. The risks of not treating the condition are much greater. It is abundantly clear that many schizophrenics do not take oral medication. Long-acting phenothiazines provide the best answer to date for this particular problem. One must hope that an over-ready acceptance of depression as a serious objection to this method of treatment does not inhibit its advance, and it need not if, as Drs. Alarcon and Carney suggest, careful supervision is maintained.—I am, etc.,

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Iatrogenic Dermatitis

SIR,—Your leading article on rosacea-like dermatitis (6 September, p. 545) warns that topical corticosteroids may worsen the condition, but there are occasions when they appear to cause it. I have described (15 March, p. 671) the rebound papulo-pustular reaction which may follow the sudden cessation of application of topical fluorinated corticosteroids in patients with rosacea. Since then I have seen similar acute papulo-pustular reactions in other conditions than rosacea, in particular in acne vulgaris.

A boy of 13, who was seen first on 5 December 1968, had suffered from acne vulgaris of his face and shoulders for a year, and for the preceding four months had applied betamethasone valerate ointment three times daily to his face. When first seen he showed the familiar telangiectasis and atrophy of the central face due to overdose of topical betamethasone. Four days after ceasing to apply the ointment his cheeks and chin became erythematous and studded with small papulo-vesicles, an appearance similar to an acute contact sensitivity. It was noticeable that the upper lip was not involved, and clinically he resembled the acute phase of perioral dermatitis. After three weeks on oral tetracycline 250 mg. twice daily and the application of hydrocortisone ointment his skin had apparently recovered.

Since the observations on rosacea, particular attention has been paid in our department to inquiry as to the local treatment used by patients with perioral dermatitis prior to their referral to hospital. All the last 10 patients with this condition seen in our department had used fluocinolone or betamethasone valerate for periods of from two months to six years. Several patients were, unknown to their own doctor, using these substances which had been prescribed for dermatoses in other members of the family and because of their magical effect had been looked on as a panacea for all ailments. It would be interesting to know whether the mother and child with perioral dermatitis reported by Verbov and Abell[1] had been using steroid creams. Professor Steigleder[2] and previous authors on this subject have not commented on this aspect of their cases. In our patients with perioral dermatitis cessation of the use of topical fluorinated corticosteroids led to a worsening of the eruption which the patient ascribed to intolerance to other applications used as substitutes, and many returned to the corticosteroids again in the same way as rosacea patients.

It may well be argued that no patient with an intractable skin eruption can now avoid treatment with potent topical steroids, but I believe that some of the increase in incidence of rosacea-like dermatitis is a result of the treatment of seborrhoeic eczema and acne vulgaris with them, and the apparent increase which has spread from America to Europe parallels the increased use of these preparations. I do accept that a papulo-pustular seborrhoeic-like eruption in the naso-labial folds as described by Frumess and Lewis[3] does exist. It was described even before triamcinolone was used, but it is a comparatively rare condition, which can be mimicked very closely by topical steroid withdrawal eruptions. Both the naturally occurring and the iatrogenic syndromes have responded fairly well to oxytetracycline given in doses of 250 mg. b.d., but only if at the same time the fluorinated steroids have also been discontinued. It may be that failure to recognize this accounts for the variable results with tetracycline reported by others.—I am, etc.,

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2 Steigleder, G. K., *Deutsche Medizinische Wochenschrift*, 1969, **94**, 1393.
3 Frumess, G. M., and Lewis, H. M., *Archives of Dermatology*, 1957, **75**, 245.

Origin of the Third Heart Sound

SIR,—I would like to amplify one point arising from the correspondence (6 September, p. 597) on the origin of the third heart sound and ventricular filling which might otherwise give rise to some confusion. Cineangiographic studies of ventricular filling show that mitral valve action is a vortex-dependent mechanism with dye circulating in the retrovalvular zones in a manner analogous to the fluid motion in the sinus of Valsava during systole. They also show the mitral valve closing *after* atrial contraction. Therefore the non-dimensional terms governing vortex formation and decay (the Reynolds and Strouhal numbers) can be applied to this situation. There will therefore be an upper limit of Strouhal number, the frequency parameter governing the time-dependent circumstances of vortex generation, and a lower limit of Reynolds number, the viscosity parameter governing the liability of a vortex to occur and persist. The duration of diastole suggests that it is the vortex decay term that is especially critical to valve closure and as a result to valve efficiency. The flatness of the ventricular filling curve after the rapid filling phase (diastasis) and then its sharp rise with atrial contraction strongly suggests that atrial systole should be seen as a means of aiding valve closure by giving the vortex an added impetus in the latter part of diastole