facts and to have an educative role. The medical profession, teachers, social workers, psychologists, and parents have responded to the challenge. Our programme is developing but we need the help of more doctors. Our brochure, "The Case for Responsibility," is available on request.—I am, etc.,

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SIR,—What a relief it is to read your leading article on the porn industry (30 September, p. 779). Would that all reasonable people make their views abundantly clear, so that action shall be taken to prevent the propagation of filth.—I am, etc.,

B. S. C. GASTER

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Iatrogenic Cushing's Syndrome

SIR,—A 42-year-old woman presented in March 1971 with a history of intermittent psoriasis for 16 years. Over the past two years it had become progressively worse and she had an almost generalized exfoliative psoriasis. The skin surface was tense, shiny, and erythematous with marked exfoliation. There were signs of follicular blockage and secondary infection. Though the skin condition certainly posed a problem, the patient's general practitioner (J.McC.) had referred her mainly on account of her Cushingoid appearance.

On questioning, it was discovered that over the past two years the patient had been applying to her total body skin surface cream containing 0.1% betamethasone 17-valerate which had been further diluted 1:8 with a bland cream. She was issued weekly with 1,000 g of the prescription and this amount (containing 125 mg betamethasone) was applied weekly between February 1969 and February 1971. On admission to hospital she had a bloated appearance with mooning of the face and marked oedema of the face and ankles. There was a tendency to bruise readily and some muscle weakness. Secondary amenorrhoea had been present since February 1971. These Cushingoid features were thought to have been caused by percutaneous absorption of the steroid preparation.

The steroid application was discontinued on admission and the initial hypertension settled in two weeks (see Table). The blood pressure has remained normal. The initial morning plasma cortisol level, though lower than usual, was not frankly abnormal. Unfortunately the evening cortisol level was not measured on admission. The initial urinary steroids were also low on admission but, like the plasma cortisol, were subsequently normal. Normal menstruation returned after three months.

When long-term application of a potent steroid preparation is stopped a rebound phenomenon takes place and the skin condition

deteriorates rapidly. In this case a dilute form of ordinary 1% hydrocortisone ointment was substituted for the betamethasone cream and applied sparingly twice daily to the lesions. Internal treatment with hydroxyurea was given under supervision. The response to this therapy was good. The exfoliation ceased and the more usual picture of localized plaques of psoriasis emerged. To date the patient has had to have intermittent courses of hydroxyurea and she continues to apply the dilute 1% hydrocortisone ointment.

The systemic effects of topical steroids have been well documented^{1,2} as producing both local Cushingoid changes in the skin and pituitary-adrenal axis suppression. These effects are dose dependent and are enhanced by occlusive dressings. The dosage of steroid cream used in this case was 18 mg/day betamethasone for about two years. Lehner and Lyne³ have shown no adrenal suppression on a topical oral dose of 0.4 mg/day, so the Cushingoid effect here is attributable to the excessive quantity of preparation used by the patient.

This case report emphasizes the hazards of long-term topical application of high-potency steroid preparations.—We are, etc.,

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¹ Fitzpatrick, T. B., Griswold, H. C., and Hicks, J. H., *Journal of the American Medical Association*, 1955, 158, 1149

² Scoggins, R. B., and Klinman, B., *New England Journal of Medicine*, 1965, 273, 831.

³ Lehner, T., and Lyne, C., *British Medical Journal*, 1969, 4, 138.

Treatment of Endocrine Impotence

SIR,—In my experience androgen therapy for "endocrine impotence" is usually unsuccessful even when the plasma testosterone level is reduced. Dr. A. J. Cooper (1 April, p. 34) called attention to the fact that there is no conclusive evidence that potency in men, once established, depends on testosterone, and Michael has reported¹ that he was unable to restore sexual behaviour to precastration levels by giving testosterone propionate. My results in treating endocrine impotence in ageing men with testosterone are disappointing. Such patients are often treated with tranquilizers or sedatives, which only mask the illness.

The testes of young, healthy men produce quite large amounts of oestrogen as well as testosterone.^{2,4} This is not a fault of nature. Most probably both oestrogen and testosterone are needed for the development and the maintenance of masculine sexual organs and their function. It is difficult to understand why these well-established facts have not been taken into consideration in hormonal therapy. Oestrogen response is

higher than androgen response to stimulation with human chorionic gonadotrophin in young healthy men, but even this finding has been ignored. I have found in 60 selected cases of male climacteric with symptoms of endocrine impotence that treatment with a mixture of oestrogens and androgens has given better results than testosterone alone. The synergistic action of the two substances in the male organism merits attention. Since 1968 I have given oestrone/testosterone parenterally in ratios of 1:10, 1:20, 1:50 together with oral oestradiol in daily doses ranging from 2 to 4 mg. In another group of elderly patients with cardiovascular disease and sexual hypofunction satisfactory results were obtained with injections of either oestrone, nicotinic acid, and testosterone (ratio oestrone to testosterone 1:20) or oestradiol and an anabolic steroid.

Improvement in sexual potency is most probably connected with vasodilatation and a better blood supply to the genital organs—that is to say, local trophicity. Oestrogens augment the blood supply to various parts of the body but primarily to the sexual organs. Further clinical trials with different mixtures of oestrogen and testosterone are needed before the best formula can be established. The advantages of a mixed therapy are: (1) it diminishes the risk of atherosclerosis and coronary thrombosis, because the oestrogenic component lessens the negative influence of testosterone on lipid metabolism; (2) it diminishes the risk of prostatic cancer; and (3) the nicotinic acid augments the lipocatabolic action and has anti-atherosclerosis properties and it causes vascular dilatation.—I am, etc.,

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Medical Academy in Warsaw,
Poland

¹ Michael, R. P., in *Endocrinology and Human Behaviour*. London, Oxford University Press, 1968.

² Michael, R. P., Personal communication.

³ Fishman, L. M., Sarfaty, G. A., Wilson, H., and Lipsett, M. B., in *Endocrinology of the Testis*. London, Churchill, 1967.

⁴ Maddock, W. O., and Nelson, W. O., *Journal of Clinical Endocrinology*, 1952, 12, 985.

Management of Diabetes Insipidus in Pregnancy

SIR,—The wide spectrum of biological activities of the neurohypophysial hormones applies also to vasopressin and can lead to undesirable complications. Thus, though there is a wide difference between the very high antidiuretic and the relatively low uterotonic actions of this hormone therapeutic antidiuretic dosages in diabetes insipidus can threaten the continuation of normal pregnancy. Recently, an analogue of the hormone, (1-deamino-8-d-arginine)-vasopressin (DDAVP), has become available,¹ and reports^{2,3} have shown that in comparison to the natural peptide DDAVP has a far higher and very prolonged antidiuretic activity with practically no other side effects on any smooth muscle. The different biological spectrum of DDAVP is a great improvement in treating pregnant patients with diabetes insipidus, as the following case illustrates:

A 30-year-old nullipara with a past history of hyperthyroidism developed acute diabetes insipidus a fortnight after a severe head injury from a car accident in the sixth month of pregnancy. Her untreated water turnover showed a

	2 March	17 March	8 April
3.P. (mm Hg)	190/120	120/80	120/80
Plasma cortisol	6 a.m. 6.9 µg/100 ml 11 p.m. —	6 a.m. 18.8 µg/100 ml 11 p.m. 11.8 µg/100 ml	9 a.m. 16.5 µg/100 ml 9 p.m. 9.5 µg/100 ml
Urinary 17-oxosteroids	3.5 mg/24 hr	5.4 mg/24 hr	6.1 mg/24 hr
Urinary 17-oxogenic steroids	4.0 mg/24 hr	8.2 mg/24 hr	12.2 mg/24 hr