gravidarum, as even in the very severe case active measures to combat dehydration and electrolyte imbalance bring about a rapid improvement in the patient's condition and one can usually tide the patients over until spontaneous remission occurs.—I am, etc.,

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Dangers of Monoamine Oxidase Inhibitors

SIR,—A number of abnormal responses to pethidine in patients receiving monoamine oxidase inhibitors have been reported. We wish to record a similar response following the use of papaveretum ("omnopon").

A 54-year-old woman weighing 74 kg. was due to have a bilateral myringotomy. For two months previously she had been treated with phenelzine ("nardil") 15 mg. t.d.s. and chlorthalidopoxide ("librium") 10 mg. t.d.s. for depression. For the last three weeks she had been receiving Gower's mixture for trigeminal neuralgia. There was a two-year history of effort angina, and during an acute episode of chest pain six months previously she had received morphine 15 mg. without ill-effect.

Papaveretum 10 mg. and L-hyoscine 0.4 mg. were given as premedication, and 50 minutes later she was found to be unrousable, peripherally dilated, and sweating. Systolic blood-pressure was 70 mm. Hg, respiratory minute volume 8.2 litres, and respiratory rate 12 per minute. The only other abnormal signs were coarse crepitations throughout the chest and a left extensor plantar response. Nalorphine 10 mg. was administered intravenously. This produced a transient rise in blood-pressure to her normal pre-operative level of 160/100 mm. Hg. There was little change in ventilation and response to painful stimuli disappeared altogether. Systolic blood-pressure fell over the next 30 minutes to around 90 mm. Hg.

Three hours after premedication, bilateral myringotomy was performed without the need for further drugs. One hour later, following the suggestion of Shee,¹ prednisolone phosphate 20 mg. was given intravenously. This produced a transient rise of systolic pressure to 120 mm. Hg and a sluggish response to painful stimuli returned. These improvements were not maintained and there was no response to a further 10 mg. prednisolone phosphate given 30 minutes later. Six hours after premedication, Valsalva's manoeuvre, recorded by an intra-arterial blood-pressure trace, showed a normal response. Seven hours after premedication she was opening her eyes and responding to her name. Her condition gradually improved and by the following morning had returned to normal.

While it is impossible to be certain that the gelsemium in Gower's mixture was not concerned in this response, we think it likely that phenelzine was responsible. The reaction was unlike simple potentiation of the action of papaveretum, as respiratory depression was not a feature. This case demonstrates that in patients receiving monoamine oxidase inhibitors abnormal and dangerous responses may not be limited to pethidine but can occur with other narcotic agents.—We are, etc.,

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REFERENCE

- ¹ Shee, J. C., *Brit. med. J.*, 1960, **2**, 507.

Chlorpropamide and Foetal Damage

SIR,—I was interested in the remarks by Dr. A. I. MacPhail, of Pfizer (January 19, p. 192), upon my observations on the above problem (January 5, p. 59). We have already noted in our paper,¹ in all fairness, that chlorpropamide has never been advocated by the company manufacturing it in the treatment of pregnancy in the established diabetic.

What I find hard to correlate with Dr. MacPhail's statement that "pregnancy is high upon the list of contraindications" for the use of chlorpropamide is the recent announcement² that his company (or its subsidiary, Messrs Pfizer Private Limited) in India has donated a "research grant of Rs.4,000" for the study of chlorpropamide in pregnant diabetics.

One of the principal purposes of my communication about high perinatal mortality was to warn any doctors taking part in the Pfizer trial in India that, in doses of 500 mg., chlorpropamide when used throughout pregnancy in insulin-independent diabetics is associated with foetal wastage. In view of our findings I would suggest, most respectfully, that it might be wise were the Pfizer research grant applied to another facet of the fascinating syndrome of glycosuria in Indians.—I am, etc.,

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Haematuria following "Jectofer"

SIR,—A 26-year-old nurse with a mild refractory anaemia for at least two years was seen at routine medical examination in May, 1962. There were no symptoms, apart from lassitude, and no abnormal physical signs at this time. She was observed and investigated in detail over the next six months. Her haemoglobin was generally about 70% (10.4 g./100 ml.), and film showed moderate hypochromasia and mild anisocytosis and poikilocytosis. She had received a course of iron dextran ("imferon") without ill effect, but had been intolerant of various oral iron preparations. Ferrous fumarate was well tolerated but failed to improve her anaemia, and a course of intravenous saccharated iron oxide ("ferrivenin") was stopped after a total of 12 ml. because of fever, sore throat, headache, and backache (possibly attributable to an intercurrent respiratory infection). It was concluded that she had a mild but refractory iron-deficiency anaemia, possibly caused by menorrhagia, though a gynaecologist was inclined to discount this as a factor.

On January 1, 1963, daily intramuscular injections of 2 ml. (100 mg.) of iron sorbitol citric acid complex ("jectofer") were commenced. Some eight hours after the fourth injection she developed frequency of micturition and frank haematuria. She was seen two days later, when there were no abnormal symptoms or signs, and a mid-stream urine showed only occasional leucocytes and red cells and was sterile on culture. After four further daily injections, however, frequency and haematuria returned together with fever and headache. The injection sites were very painful. Her urine was sterile but contained albumin and numerous red cells. After

four days' rest all abnormal features subsided. Subsequently an intravenous pyelogram showed normal appearances of kidneys, ureters, and bladder; and her blood urea was 22 mg./100 ml. Hb rose to 84% (12.4 g./100 ml.).

There have been several published reports on the use of iron sorbitol citric acid complex. Andersson¹ reported favourably on its use in 39 cases, only mild local side-effects being noted. Scott,² however, reported three moderately severe reactions—two patients had headache and vomiting two hours after injection, and a third patient experienced dizziness and disorientation. It is also interesting to note that urinary symptoms developed next day in one of the patients (apparently due to a coliform infection). It was postulated that the concurrent administration of oral iron might be responsible, but Kinloch³ later reported similar reactions together with a metallic taste in the mouth in four patients who were not receiving oral iron.

This patient was not receiving oral iron at the time of her haematuria. It is known that iron sorbitol citric acid complex is rapidly absorbed, some 70% being removed from the injection site within three hours. No effect on the human blood coagulation time has been observed. About 30% injected iron is excreted in the urine in 24 hours, most within the first few hours.⁴ No adverse reports on renal function have been made hitherto, but the relation between the injections of jectofer and the appearance of haematuria in the present case cannot be ignored. Opinions on the lack of serious toxic side-effects of this preparation should therefore be revised, and its future administration considered in the light of this report.—I am, etc.,

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- ¹ Andersson, N. S. E., *Brit. med. J.*, 1961, 2, 275.
- ² Scott, J. M., *ibid.*, 1962, 2, 480.
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Psychiatrists and Patients

SIR,—In discussing the team system and admissions to a mental hospital (February 9, p. 389) Drs. G. Milner, K. Kumar, and A. H. Bakkar perform a valuable service in drawing attention to the different kinds of patients who come under the care of the particular psychiatric consultants working at their hospital. Recently I examined the proportion of new men and women out-patients referred during 1962 to three particular psychiatrists at another hospital.

	Psychiatrist								
	A			B			C		
	M	F	M/F × 100	M	F	M/F × 100	M	F	M/F × 100
New out-patients ..	33	53	62	80	75	107	60	35	171
Admissions ..	47	83	57	60	72	83	94	82	115

More women than men were referred to psychiatrist A, roughly equal numbers to B, and an excess of men to C. The variation in sex ratio of admissions was rather less but still in the same direction.

Some years ago¹ I described the significant difference in the somatotype distributions of two groups of undergraduate patients (men), the one group with an excess of more muscular builds who had come under the care

of a woman psychotherapist, the other with an excess of fatter physiques under the care of a male psychiatrist. The distribution is set out in the following Table:

	Somatotype Dominance			
	Musc.	Lm*	Lf*	Fat
Woman psychotherapist ..	6	17	11	2
Male psychiatrist ..	16	13	27	21

$$\chi^2_{18} = 14.4$$

* Linear builds: secondary dominance, Lm = muscular, Lf = fat.²

Further notes were given of significant somatotype differences in the strength of the counter-transference which developed and in the recorded outcome of treatment. There would be little point in trying to compare the effectiveness of the two psychotherapists, who were treating different kinds of undergraduates, yet this is the kind of comparison commonly made when results of therapeutic trials, even double blind trials, are blindly compared. Insufficient effort has been made in most, if not all, "controlled" trials to match for such obvious and measurable features as physique, the important effects of which on prognosis are only now being adequately measured.³—I am, etc.,

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- ¹ Parnell, R. W., Chapter in *The Nature of Stress Disorders*, 1958. Hutchinson, London.
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Oranges and Lemons

SIR,—Having served in the R.A.F.M.S. during the war I have a high regard for Sir Geoffrey Keynes, but I cannot allow him (February 16, p. 475) and Pertinax (February 23, p. 533) to accord to John Woodall the credit of being the first to advise orange and lemon juice for the treatment of scurvy.

The first sentence in Lind's *Treatise of the Scurvy* (1753) reads, "In the first accounts given us of this disease, by Ronsseus, Echthius, and Wierus, it is surprising to find, not only an accurate description of it, but an enumeration of almost all the truly antiscorbutic medicines that are known to the world even at this day."

These three authors wrote between 1541 and 1567. Ronsseus (1564) wrote that seamen in long voyages cure themselves of it by the use of oranges. Lind's controlled experiment was necessary because the work of Eugalenus Severinus¹ brought confusion to the diagnosis of scurvy and discredit to the use of antiscorbutics. His book ran through a number of editions between 1604 and 1720, and was recommended as the standard work on scurvy by the great Boerhaave.

In spite of the controlled experiment of Lind, it took the Lords of Admiralty 40 years to give effect to his recommendations. In spite of Lind's work Garrod² claimed that by adding 12–20 grains (0.78–1.32 g.) of potash to the scorbutic diet he could cure his patients as quickly as by the addition of fruits and vegetables: Ralfe³ showed that the primary alterations in scurvy seemed to depend on a general alteration between the various acids and bases found in the blood; Almroth Wright⁴ taught that in scurvy there was a diminished alkalinity of the blood and that scurvy could be prevented and cured by the administration of calcium lactate; and in 1912 P. O. E. Evans and Captain L. Oates died of scurvy on the march back from the South Pole.