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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REFERENCES

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² Fairweather, D. V. I., and Loraine, J. A., *Brit. med. J.*, 1962, **1**, 666.

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

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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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- ¹ Jackson, W. P. U., Campbell, G. D., Notelowitz, M., and Blumsohn, L., *Diabetes*, in press.
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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