Depressive Illness in General Practice

SIR,—It is with interest one reads the paper by Dr. A. M. W. Porter on depressive illness in a general practice (28 March, p. 773). While Dr. Porter has put a lot of hard work into his paper, I feel it would have been valuable to enlarge on the parameters of the rating scales in order to bring out the important clinical assessment of depression such as external stress factors (reactive) and whether the depression is worse in the mornings. Is there sleep disturbance? Are there associated guilt feelings? Is there weight loss and poor appetite, and, most important, were there associated suicidal feelings? Stressing these clinical aspects would, I feel, have added more evidence to his conclusion.

I think it would also have been helpful to know what part psychotherapy played in the treatment and assessment, as it is important not to dissociate medication from psychotherapy even in clinical trials.

I feel it is a drawback that the duration of the trial was three weeks only. The conclusions arrived at might have been more significant if the duration of the trial was more than three weeks, since the Medical Research Council trial[1] showed that imipramine is a very worthwhile drug for treating depression. Dr. Porter indicated in his paper that in general practice quick results are expected. I think one would agree with him on this point so far as treatment of organic illness is concerned, but, with emotional illness, it takes certainly longer in view of the interactions of personalities and adaptation to stress factors, psychotherapy, etc.—I am, etc.,

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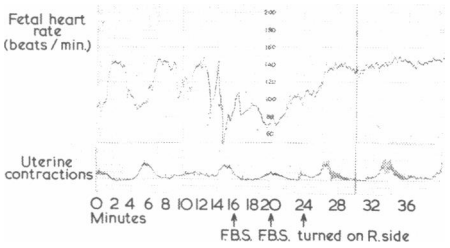
REFERENCE

1 Medical Research Council, *British Medical Journal*, 1965, 1, 881.

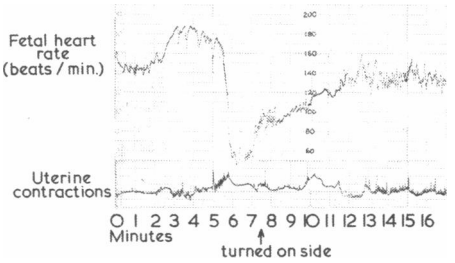
Supine Hypotension Syndrome

SIR,—Dr. L. Courtney (28 March, p. 797) has drawn the attention of obstetricians and anaesthetists to the risks of the supine hypotension syndrome to the mother at caesarean section. The death of one of the patients whose case history he describes underlines this point. Yet surely the adverse effects of supine hypotension on the fetus deserves some mention. It is likely that with either the fall in cardiac output or peripheral vasoconstriction that accompanies the condition there is a diminution in blood flow through the intervillous space. The extent to which uterine blood flow is affected is not known, but there is little doubt that fetal hypoxia is a common sequel.

During the course of continuous monitoring of the fetal heart rate in our intensive care labour ward at King's we have recorded acute fetal distress in five patients in the supine position, which was relieved by turning the patient on her side. The rapid improvement that accompanies this procedure is shown in the continuous fetal heart records obtained from two patients.



The patient whose record is shown in Figure 1 was known to suffer from supine hypotension, having had two attacks when placed in the lithotomy position prior to artificial rupture of the membranes. When labour started she was turned on to her back so that the midwives could record the fetal heart rate. Although she disliked this position, she was able to tolerate it. An irregular fetal heart rate was reported so a scalp electrode was applied to the fetus. The record shows an irregular bradycardia lasting 30 minutes. The *p*H of two fetal blood samples (F.B.S.) taken at this time was 7·20 and 7·18. The arrow shows the point when the patient was turned on her side, and an immediate recovery of the fetal heart rate to a normal level can be seen. The fetal *p*H 30 minutes later was 7·26.



The record in Figure 2 shows the change in fetal heart rate which was not accompanied by changes in fetal *p*H during a clinically more acute but shorter episode of supine hypotension than in the previous case. The patient, who was in the supine position, complained of feeling faint, became pale, and started to sweat. She recovered quickly as soon as she was turned on to her side. Figure 2 shows an episode of fetal tachycardia lasting three minutes followed by two minutes of profound bradycardia relieved by turning the patient on to her side.

Our experience of the beneficial effect to both mother and fetus of turning the mother on her side has encouraged us to try this simple expedient in all cases of fetal distress during labour. If, as a consequence, there is an improvement in the fetal heart rate pattern and *p*H labour is allowed to continue.—We are, etc.,

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Oral Lignocaine

SIR,—The paper by Dr. P. I. Parkinson and others (4 April, p. 29) was of interest to us as we have also carried out studies on the oral absorption of lignocaine in healthy volunteers. We are most surprised by the very high blood levels of lignocaine reported by these authors, as such levels are unobtainable by similar dosages given by any other route, including intravenously. In the cases studied by us[1] 500 mg. orally gave maximum plasma levels of 1-2 μg./ml.—that is, one tenth of those reported by Dr. Parkinson and others. Plasma levels were were determined by gas chromatography and were cross-checked by two independent laboratories, both with an extensive experience of plasma lignocaine estimations. Furthermore, the lignocaine blood levels in excess of 10 μg./ml. reported by Dr. Parkinson would be expected to give rise to convulsions or even coma.

We also noticed transient symptoms of toxicity in volunteers receiving oral lignocaine. These occurred at much lower plasma levels than has been observed with intravenous administration. We suggested that in its passage through the liver immediately following absorption the lignocaine was partially metabolized and that the metabolite was responsible for the toxic effects.

The anti-arrhythmic effects of oral lignocaine, reported by Dr. Parkinson and colleagues, are of interest, although it is extremely difficult to evaluate the anti-arrhythmic effects of orally administered drugs. If their observations can be confirmed, it suggests to us that the effectiveness of lignocaine by the oral route may be greater than can be accounted for by the plasma levels of lignocaine achieved. It is possible that such an anti-arrhythmic effect could be achieved by a metabolite of lignocaine.—We are, etc.,

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REFERENCE

1 Scott, D. B., Jebson, P. J., Godman, M. J., and Julian, D. G., *Lancet*, 1970, 1, 93.

Mefanamic Acid-induced Haemolytic Anaemia

SIR,—Dr. G. L. Scott and others[1] have reported three cases of haemolytic anaemia in association with mefanamic acid (Ponstan) therapy. The mechanism of red cell destruction appeared to be autoimmune in nature, similar to that described for methyldopa. The subject of immune drug-induced haemolytic anaemia has recently been reviewed by Worrledge.[2] We report here haemolytic anaemia occuring in a patient with rheumatoid arthritis who was being treated with mefanamic acid, gold, and prednisone. An immune aetiology could not be detected.

The patient was a 50-year-old Caucasian male who had had rheumatoid arthritis for eight years. His previous drug treatment consisted of phenylbutazone, indomethacin, soluble aspirin, hydroxychloroquine, chloroquine, prednisone, and gold. In March 1967 he was receiving 10 mg. prednisone daily, and to this was added intramuscular sodium aurothiomalate (Myocrisin) every three weeks together with mefanamic acid (1-1·5 g. daily). This treatment was continued until his admission to hospital on 28 June 1969 with complaints of giddiness, tiredness, shortness of breath on exertion, nausea, and occasional vomiting. Haemoglobin was 7·4 g./100 ml.,