

areas—notably those heavily industrialized and heavily populated—where a hospital-confinement rate higher than 70% is desirable. It is in those areas presumably that the effects of the expected increase in the birth rate will also be more keenly felt and the evil effects of too early discharge most likely to be seen, and, as Sir Andrew warns us, “do not let us therefore imagine that it is the ideal arrangement.”

From the beginning, those most earnestly advocating this makeshift have been obsessed with only one aspect of its value, and, inasmuch as it is aimed at reducing the risks of labour itself, it obviously serves its purpose. When labour is successfully ended there are two individuals for whose welfare we and our colleagues, the paediatricians, are jointly responsible, and in the area in which I work our consultant paediatrician has expressed his concern at babies being discharged on the second or third day. For this and a number of other reasons it is proposed to conduct a “paper exercise” during ensuing months by discharging patients about the sixth or seventh day. This seems a reasonable compromise, and in some areas may enable all reasonable needs to be met without great strain on nursing and other resources. Nor does it seem unreasonable to suggest that many women nowadays, in better general health, well-prepared for labour during a properly conducted pregnancy, may be expected to tolerate a shorter stay in hospital after delivery. Furthermore, it seems questionable whether all regions will encounter to the same extent the anticipated rise in the birth rate forecast for the next few years. After all, your leading article (31 October, p. 1089) informs us that now in Britain the number of women taking the “pill” may amount to nearly a quarter of a million—who knows?—I am, etc.,

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H. VINCENT CORBETT.

### Age for Cervical Cytology

SIR,—Your *Supplements* of 17 and 31 October (p. 156 and p. 165) state that women between the ages of 35 and 50 years should be accepted for screening by cytology. These ages are quite unrealistic.

In 150 consecutive cases of invasive carcinoma of the cervix I found the ages to be: 20's 3; 30's 17; 40's 33; 50's 51; 60's 32; 70's 13; 80's 1. The average age for carcinoma-in-situ is about 13 years earlier. The age at which carcinoma-in-situ becomes common in our cases is 28 years. When all women at risk have been screened earlier there will be little point in repeating these examinations after the age of 60 years; to stop at 50 now would be to miss most of the cases. In the past year, from the antenatal clinic, we have detected four cases of carcinoma-in-situ between the ages of 16 and 18.

The service must be available to all women at risk who wish to avail themselves of it. The difficulties and expense of providing this service are grossly exaggerated. Our service in diagnostic cytology is now 16 years old; it will cope with any demands made on it even if the 170,000 women at risk in the area are all tested in the foreseeable future.—I am, etc.,

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### Fifty Years On

SIR,—In Sir Macfarlane Burnet's highly stimulating article on medical education, “Fifty Years On” (31 October, p. 1091), he designates five areas of study and describes four types of doctors. He then allocates various weightings of intensive study for these different types of doctor. Each undergraduate is presumably to share a common pathway of medical education, indicated by one +, with elective courses of different intensity indicated by two ++ or three ++++. Many of us must be coming to a similar conclusion that these different pathways of medical education are inevitable. The main problem is the timing of the elective courses. Most undergraduates do not decide where their vocation lies until they come to the end of their clinical course. If the elective courses are to be offered in the pre-clinical period how shall we decide which students should enter them? And alternatively if we decide that these elective courses should be postponed beyond the qualifying examination shall the common pathway (+) for all undergraduates last no more than three or four years?—I am, etc.,

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DOUGLAS HUBBLE.

### Low Molecular-weight Dextran for Foetal Distress

SIR,—While it may be that the use of intravenous low-molecular-weight dextran (Rheomacrodex) is of value in the treatment of foetal distress in labour I cannot accept that this has been convincingly shown by Mr. J. B. Jones in his article (10 October, p. 909).

Foetal tachycardia (160/min. or over) is not always an index of distress as is foetal bradycardia, and without the added sign of meconium it is not necessarily indicative of foetal anoxia. It is my experience that one of the commonest causes of foetal tachycardia in labour is maternal ketosis, which can be countered with intravenous dextrose infusion, with early return of the foetal heart rate to normal levels. The majority of Mr. Jones's cases were foetal-tachycardia cases, and I feel that it is not justifiable to conclude that the low molecular-weight dextran in the Rheomacrodex/dextrose solution was responsible for the “improvement” in the foetal state. Furthermore, in cases of foetal bradycardia the state can be temporary while a head has “moulded through” a tight plane of the pelvis, or descended suddenly from abdomen to floor of pelvis.

If a larger series is to be completed to give full assessment of value I hope that consideration shall be given to points which I have made, particularly the maternal pulse rate and ketosis, and the incidence of passage of meconium. At present it would seem that oxygen administration is first choice as an emergency measure while arrangements are being made to expedite delivery.—I am, etc.,

Walsall, Staffs.

CALUM N. MCFARLANE.

### Oral Contraception and Liver Damage

SIR,—I have read with interest your correspondence relative to liver toxicity result-

ing from oral contraceptive pills. In one instance, as reported by Dr. A. Eisalo and his colleagues (15 August, p. 426), each of seven patients were said to have hepatic toxicity as a result of taking Lyndiol (5 mg. 17 $\alpha$ -ethinyloestrenol and 0.15 mg. 3-methoxy-17 $\alpha$ -ethinyloestradiol) and in another (Drs. I. P. Palva and O. O. Mustala, 12 September, p. 688) five consecutive postmenopausal patients demonstrated “hepatic toxicity” on Anovlar (4 mg. norethisterone acetate and 0.05 mg. of ethinyloestradiol).

I would like to call attention to my report in the *B.M.J.* (3 October, p. 843) which failed to show any similar occurrence of abnormal liver function tests in studies of hundreds of patients. As I indicated in the report, I am sure there is more to this particular problem than abnormal laboratory findings when and if they occur.

If the findings in the small series were to be transposed to the millions of women now taking oral contraceptive pills for several years, it would seem that by this time hundreds would have shown frank evidence of liver pathology. If it is argued that the age-group of patients in the two very small-scale studies might result in results different from those among the younger patients taking oral contraceptive women on another leave “no doubt see more severe liver pathology among patients who are using the identical hormones in treatment of the menopause. Certainly, ethinyloestradiol, the oestrogenic component of Anovlar (and the authors have suggested that the oestrogen is the substance incriminated), has been given to hundreds of thousands of women receiving menopausal treatment.

I think Drs. Palva and Mustala are somewhat rash in stating that the results of experiments on seven women on one compound and five women on another leave “no doubt about the hepatotoxicity of this contraceptive pill.” We can agree with their statement that further studies are indicated, as we emphasized in our own clinical report, but believe the authors are ignoring the very extensive amount of work that has already been done in our laboratories and elsewhere, and the fact that laboratory tests, *per se*, are not enough to prove a clinical diagnosis.—I am, etc.,

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EDWARD T. TYLER.

SIR,—The problem of oral contraceptives and liver damage has been the subject of the preliminary communication of Drs. A. Eisalo and his colleagues<sup>1</sup> and some letters in your journal.<sup>2-4</sup> The series of Eisalo and his colleagues and Palva and co-workers<sup>3</sup> consisted of postmenopausal women and showed elevation of the values for serum glutamic pyruvic transaminase and serum glutamic oxaloacetic transaminase. On the other hand, Dr. Swaab<sup>4</sup> found no signs of hepatic injury during oral contraceptive medication in a series of young women. We have recently observed a patient with severe intrahepatic cholestasis during treatment with oral contraceptives.

The patient was a woman, aged 24, with a history of epidemic hepatitis at the age of 10. During two normal pregnancies, in 1961 and in September 1963, no signs of prurigo or jaundice were noted. She denied having taken any drugs