

the sample as a whole may not reflect the general view, but it is possible that it does do so.

¹ Committee on Privacy (Chairman, K. Younger), *Report*, Cmnd. 5012. London, H.M.S.O., 1972 (£2 net).

² British Association of Social Workers, Working Party on Confidentiality in Social Work, *Discussion Paper No. 1, Confidentiality in Social Work*. London, B.A.S.W. Publications Dept., 1971.

³ *British Medical Journal*, 1971, 4, 316.

⁴ *British Medical Journal Supplement*, 1972, 2, 108.

Oral Contraceptives Containing Only Progestogens

Oral contraceptives containing only a progestogen have had a chequered career. In 1965 trials in Latin America showed that very small amounts of a progestogen, chlormadinone acetate, when given without any added oestrogen had a strong antifertility effect. Not all progestogens are suitable for use alone. Indeed with norethynodrel, the progestogen most commonly used in the late 1960s, the amount of oestrogen was critical at low dosage.¹

The preliminary trials in Britain of oral contraceptives containing only progestogen were encouraging, and when it became evident that the thromboembolic deaths associated with oral contraceptives could be attributed to the oestrogenic component it seemed that the progestogen-only pill might in many cases be an acceptable alternative. These hopes were dashed when Eleanor Mears and colleagues² did a clinical trial of four progestogens in Yugoslavia. They found an appreciable failure rate with megestrol acetate, chlormadinone, norgestrel, and norethisterone, and concluded that continuous administration of small doses of progestogen were unlikely to offer a serious challenge to the existing oestrogen-progestogen mixtures. Now that Professor P. Eckstein and his colleagues have returned to the charge with a clinical trial of norgestrel published in this issue of the *B.M.J.* (page 195), it is justifiable to consider this compound anew.

The differences in the findings on norgestrel by Professor Eckstein and his colleagues and those of the earlier study by Eleanor Mears and her colleagues are more apparent than real. It is true that in the earlier study norgestrel was associated with a pregnancy rate of 4 per 100 woman-years while the worst interpretation of Professor Eckstein and colleagues' data gives a pregnancy rate of 2 per 100 woman-years, but in the earlier study a lower dose of norgestrel (50 µg) was used than in the present one (75 µg). One conclusion is inescapable: there is a small but indubitable failure rate with norgestrel, but when the mixed oestrogen-progestogen pills are taken correctly the pregnancy rate is virtually nil.

The subjective side effects of oral contraceptives, such as headaches, lassitude, and changes in libido, are notoriously difficult to assess, and different observers have found widely different incidences of such symptoms with the same pill. It is a happy chance that the Birmingham group which took part in the present trial on norgestrel have also published their findings on the side effects experienced by patients taking a mixture of norethynodrel and mestranol.³ There does not appear to be any serious difference between progestogen-only and progestogen-oestrogen mixtures in this respect, and it is fair to compare the two forms of contraception in terms of their effectiveness, their effect on the menstrual cycle, and their tendency to cause thromboembolism or affect carbohydrate metabolism.

It is in respect of their effect on the menstrual cycle that progestogen-only contraceptives compare most unfavourably with progestogen-oestrogen mixtures. It is true that the latter tend to prolong the cycle, and amenorrhoea after coming off the pill is now a common and troublesome finding, but these disadvantages are outweighed by the frequent and irregular bleeding often associated with progestogen-only pills. In the present study on norgestrel 20.6% of all cycles lasted less than 17 days, and a large proportion of the patients in the trial found this unacceptable.

Progestogen-only pills are less efficient than oestrogen-progestogen mixtures because they have only part of the range of activities of the latter. Thus oestrogen-progestogen mixtures inhibit ovulation, render the cervical mucus impenetrable to sperm, affect transport of the ovum in the Fallopian tube, and diminish the receptivity of the endometrium to implantation. On the other hand the contraceptive effect of progestogen-only pills depends mainly on their effect on cervical mucus. Eckstein and colleagues suggest that norgestrel may also affect the capacity of the corpus luteum to produce progesterone, but an alternative interpretation of their findings is that ovulation was inhibited in some patients. Inspection of their pregnanediol results shows that the low mean pregnanediol excretion during the luteal phase of their treated patients could be due to the inclusion of a number of patients excreting non-ovulatory amounts of the steroid. Those who did ovulate excreted amounts of pregnanediol well within the normal range of the luteal phase. It has been suggested on other grounds—for example, endometrial biopsies—that progestogens can inhibit ovulation in some patients. This is likely to be a dose-related phenomenon, and Eckstein and colleagues were using doses higher by half than those usually given.

Progestogen-only oral contraceptives are slightly less efficient than the oestrogen-progestogen mixtures. They often cause irregular bleeding at frequent intervals. They cause less endocrine alteration and probably do not have the dangerous effects of oestrogen on venous thrombosis and carbohydrate tolerance. How the sum is added up for each patient is a matter of individual judgement. The role of progestogen-only oral contraceptives is much smaller than that of the established combinations, but until means of contraception which are less of a physiological intrusion become available the progestogen-only pill has a useful function in those patients for whom oestrogen is contraindicated or who are intolerant of the mixed pill.

¹ Mears, E., *British Medical Journal*, 1961, 2, 1179.

² Mears, E., Vessey, M. P., Andolsek, L., and Owen, A., *British Medical Journal*, 1969, 2, 730.

³ Eckstein, P., et al., *British Medical Journal*, 1961, 2, 1172.

Contaminated Infusion Fluids

The fact that the Clothier report¹ contains little information that will be new to readers of the national press is a compliment to the scrupulous and open way in which the inquiry was conducted and to the high standard of its reporting. About one-third of the sub-batch of bottles of 5% dextrose infusion fluid associated with the incidents at Devonport in early March 1972 failed to reach sterilizing temperature, because of retention of air within the autoclave throughout the sterilizing cycle. Evidence of this failure was given by the recording thermometer failing to indicate any rise in temperature, but this warning was ignored, not for the