sulphamethoxazole mixture is a drug of first choice for this condition, but there are others which are equally useful.

Trimethoprim is teratogenic in rats, and although it does not have the same effect in the rabbit there can be no guarantee that it will never damage the human fetus. Since it seems likely that the effect is through interference with fetal folate metabolism—and rare cases of impaired folate metabolism have already been recognized in adults taking trimethoprim—there is some slight reason to suppose that the human fetus may be more rat-like than rabbit-like. The above information is all part of the “full information supplied on request” by the manufacturers of both Septrin and Bactrim, but the average doctor contents himself with a much smaller amount of reading material which can only contain the hackneyed phrase “contraindicated in pregnancy” common to nearly every new drug.

I have talked to many doctors in general practice and in teaching hospitals. Almost all have used trimethoprim/sulphamethoxazole mixture. None of them have specifically known this risk. Even a recent textbook on antibiotics omitted the information. One can only presume that “wolf, wolf” has been cried once too often.

Professor W. Brumfitt in the August issue of the *Prescribers' Journal*¹ states the risk, but suggests that the drug combination is suitable therapy for the bacteriuria of pregnancy. He also states that “so far there is no evidence of any adverse effect on the human foetus.”

Experience with thalidomide has made us aware of the hazards of the use of drugs in pregnancy, but has also led to the assumption that fetal abnormalities induced by a particular drug will be easily recognizable because they follow a consistent pattern and will probably be of unusual type. This is not necessarily going to be the case next time. Since the usual incidence of human fetal abnormalities is about 2% of live births, it would be necessary to give trimethoprim/sulphamethoxazole mixture to a large number of pregnant women in a prospective trial in order to establish that it produces no increase in the above figure. This trial would obviously be unethical.

For the moment trimethoprim/sulphamethoxazole mixture should only be used in pregnancy if there is no suitable alternative. With the modern therapeutic armamentarium this should be a rare event, but if the Royal College of Obstetricians and Gynaecologists were to provide a centre for the prospective reporting of all instances in which trimethoprim/sulphamethoxazole mixture had been used in pregnancy, an acceptable statistical answer might be available within a few years.—I am, etc.,

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¹ Brumfitt, W., *Prescribers' Journal*, 1971, 11, No. 4, 94.

Chorea and the Oral Contraceptives

SIR,—I read with interest the latest report of oral-contraceptive-related chorea by Dr. D. Riddoch and others (23 October, p. 217) and their comment on the absence of previous episodes of chorea or rheumatic fever in five of their six cases. Similar absence of past history was also my experience in

Canada with a further case,¹ where the development of hemichorea in a young woman afforded the opportunity of recording her hand and wrist movements by sequential and multiple exposure photography. This 20-year-old, nulliparous married woman developed hemichorea after taking an oral oestrogen-progestogen preparation for three months. Immediate improvement followed withdrawal of the hormonal agent and the institution of oral penicillin treatment; within 10 weeks the movements had entirely disappeared.

Dr. Riddoch and his colleagues achieved complete remission of symptoms by withdrawal of the oral contraceptive alone, but I would submit that we are not yet in a position authoritatively to exclude penicillin from the treatment of oral-contraceptive-related chorea. Its administration in classical Sydenham's chorea is intended to prevent relapses of the chorea and valvular involvement consequent upon the carditis, which may often be clinically silent.² In children it is well recognized that chorea may be the sole manifestation of acute rheumatic fever,³ and in a study of 1,033 children⁴ chorea alone was the clinical feature of the first attack in 12%. Evidence for supposing that lone non-hereditary chorea in the adult can be a rheumatic manifestation is analysed by Paradise,⁵ who shows that, although such diseases as systemic lupus erythematosus may be responsible, there is still a sound basis for believing that clinically isolated chorea is often due to acute rheumatic fever. The validity of a clinical diagnosis of rheumatic chorea in the absence of cutaneous, joint, cardiac, serological, or haematological abnormality seems well accredited.^{2,5}

Furthermore, in the absence of convincing neuro-anatomical proof it seems improper to highlight vascular mechanisms in discussing the pathogenesis of chorea associated with oral contraceptives, as, for example, one author⁶ has assembled references to more than 40 illnesses in which chorea has been observed including encephalitides, febrile exanthemata, arteritides and other vascular lesions, degenerative disorders, industrial and drug intoxications, drug reactions, vitamin deficiencies, and metabolic and endocrine disturbances.

In seeking the proximate cause for oral-contraceptive-related chorea, we must surely eschew an unjustifiably simplistic view of cerebral function in vascular/structural terms. As for the place of penicillin in treatment the question hinges upon whether or not there is causal relation with streptococcal infection, as in acute rheumatic fever. Answers will come only through careful follow-up of these cases as countrywide (or even international?) interest brings more to light.—I am, etc.,

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¹ Malcolm, A. D., and Howell-Jones, M., *Medical and Biological Illustration*, 1971, in press.

² Thiebaut, F., *Handbook of Clinical Neurology*, Vol. 6, ed. P. J. Vinken and G. W. Bruyn, p. 409. Amsterdam, North Holland Publishing Co., 1968.

³ Wannamaker, L. W., in *Textbook of Pediatrics*, ed. W. E. Nelson, V. C. Vaughan, and R. J. McKay, p. 533. Philadelphia, Saunders, 1969.

⁴ Perry, C. B., *Annals of the Rheumatic Diseases*, 1969, 28, 471.

⁵ Paradise, J. L., *New England Journal of Medicine*, 1960, 263, 625.

⁶ Greenhouse, A. H., *Archives of Internal Medicine*, 1966, 117, 389.

Reversible Sterilization in the Female

SIR,—Mr. E. A. Williams's letter (30 October, p. 297) on this topic is of paramount importance at a time when all gynaecologists are being asked to perform more and more sterilizations in their patients. For some years I have been using the method of diathermy coagulation owing to the ease, speed, and what I feel must be its great effectiveness, and the facility with which this can be done via the laparoscope is adding enormously to the popularity of this method, with both gynaecologist and patient. Other correspondents have also emphasized the increasing number of young patients requesting sterilization, and the time must inevitably come when a fair sprinkling of these young mothers may have cause to regret their insistence in asking for this operation. As Mr. Williams points out, if their tubes have undergone diathermy coagulation there will be so little viable tube left that reconstitution will be out of the question.

I agree with Mr. Williams that so long as there is little loss of tube, however, there should be a reasonably good prospect of anastomosing the ends together. Many years ago a patient in whom I had removed one appendage for a large ovarian cyst, and had at the same time dissected a fimbrial cyst from the other broad ligament, was later found on salpingography to have developed a stricture in the one remaining tube. This stricture was excised and end-to-end anastomosis carried out and pregnancies soon followed. Although I am normally reluctant to consider total surgery in infertility, I suspect that the overall poor results of these operations are due to the damage sustained by the tubal mucous lining by previous inflammation. If there has been no infection in the tube, however, so that its ciliated epithelium is working normally, the prospect of success should be greatly enhanced.

It is, therefore, my practice, where the possibility of reconstitution might arise, to carry out the sterilization by dividing and ligating the tubes near to the junction of isthmus with ampulla and opposite an avascular window in the broad ligament. With mosquito forceps the layers of the broad ligament are then separated and the ends of the ligated tubes buried in this space with a fine atraumatic suture. Though I have not yet been requested to reconstitute tubes after this operation, I feel that if this should happen there should be reasonable hope of success.—I am, etc.,

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Aetiology of Bell's Palsy

SIR,—Your recent leading article on “Aetiology of Bell's Palsy” (2 October, p. 2) fails to mention two specific conditions connected with increased frequency of peripheral facial weakness. One is hypertension, and you dedicated a leading article to this connexion five years ago (24 December 1966, p. 1547). It is more than a century now since W. Moxon showed pathological evidence of haemorrhage into the fallopian canal in a man with Bright's disease (and presumably hypertensive) with facial weakness. The connexion between hypertension and Bell's palsy has been demonstrated repeatedly, particularly in children. In a series of 130 patients seen here hypertension existed in 21 (16%),