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.,

L. M. McEWEN

St. Mary's Hospital Medical School,
London W.2

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.,

ALASDAIR D. MALCOLM

Division of Medicine,
London Hospital,
London E.1

- 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.,

A. L. DEACON

Birmingham 15

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%),

all adults. It is likely that ischaemia, rather than haemorrhage, was the more frequent pathogenic mechanism.

The other disease which you did mention is diabetes mellitus. Diabetes, like hypertension, is a predisposing factor. In large series the frequency of previously recognized diabetes was 10-14%.^{1,2} However, when glucose tolerance tests are performed many new cases are discovered, the frequency of abnormal results increasing with age.¹ The frequency of overt and latent diabetes in patients with Bell's palsy is thus similar to that previously reported in oculomotor palsy.³ Patients with Bell's palsy frequently show abnormal blood vessels in their conjunctivae and nail-beds, and I have therefore postulated that diabetic microangiopathy may be a factor responsible for the nerve lesion.

Your leading article mistakenly attributes to Dalton the evidence that herpes zoster infection can cause Bell's palsy. Ramsay Hunt suspected this possibility when he described the syndrome which now bears his name, and it was Aitken and Brain⁴ who demonstrated high titres against the virus in some patients with typical Bell's palsy without vesicles. Though the possibility of an unrecognized viral infection causing Bell's palsy is interesting most patients lack any evidence of such a preceeding or concurrent infection. I have tried to isolate viruses from patients with Bell's palsy and also to detect antibodies to several viruses, but the results have been negative thus far.

The seasonal variation in the incidence of Bell's palsy which is frequently mentioned and which you quote is of course only circumstantial evidence that the disease is infective. As Leibowitz⁵ remarked, similar climatic influences have been shown on the occurrence of myocardial infarction (and also cerebral vascular disease). Leibowitz has subsequently demonstrated⁶ (and not in the paper you mistakenly quoted) clustering of cases of Bell's palsy. This interesting finding is open to similar alternative interpretations.

A final point concerns the theory that water retention is a predisposing factor in the development of Bell's palsy. I have found that pregnant women are more susceptible to Bell's palsy than expected, particularly just before delivery, and also in the puerperium.⁷ Water retention could be a factor there too.—I am, etc.,

AMOS D. KORCZYN

Beilinson Hospital,
Tel-Aviv University Medical School,
Petah Tikva, Israel

¹ Korczyn, A. D., *Lancet*, 1971, 1, 108.

² Hauser, W. A., Karnes, W. E., Annis, J., and Kurland, L. T., *Mayo Clinic Proceedings*, 1971, 46, 258.

³ Korczyn, A. D., *Lancet*, 1971, 1, 862.

⁴ Aitken, R. S., and Brain, R. T., *Lancet*, 1933, 1, 19.

⁵ Leibowitz, U., *Neurology (Minneapolis)*, 1966, 16, 1105.

⁶ Leibowitz, U., *Brain*, 1969, 92, 109.

⁷ Korczyn, A. D., *Acta Neurologica Scandinavica*, in press.

No Smoking and Life Expectancy

SIR,—In recent months the *B.M.J.* and numerous others have discussed the dangers of smoking. Everywhere, however, there appears one great omission. In its anxiety to persuade the public to abandon tobacco and the consequent illnesses and disability produced as a late penalty, it is important that the medical profession should also be far

sighted beyond a mere clinical field. It is not sufficient to abolish an undesirable disease if, as a consequence, more misery is produced later. A parallel situation has occurred in recent years where the prevention of malaria by removing an agent of population control has led to widespread starvation.

When the effects of reduced smoking bear fruit, we shall have an increased life expectancy and a larger number of elderly citizens. The people who have smoked are at present likely to die at an age when the younger generation is finding them a burden which they are unwilling to bear. We must therefore couple with our exhortations for the abolition of smoking a call for advanced creation of suitable homes and geriatric services to cope with the new population bulge that will develop.—I am, etc.,

GEORGE T. WATTS

General Hospital,
Birmingham

Diagnosis of Cystic Fibrosis

SIR,—I would like to comment on Dr. A. G. F. Davidson and Professor Charlotte M. Anderson's (6 November, p. 362) reference to the screening of meconium for the presence of albumin.

They state, quite correctly, that 10-15% of patients with cystic fibrosis have no clinical evidence of malabsorption and that such cases may not show a positive meconium test.

I mentioned earlier (11 September, p. 639) the routine screening programme being carried out in Newcastle using this technique. One of the aims of this experiment is to find out how many cases of cystic fibrosis are missed for this, or any other reason.

We do not know as yet if absence of malabsorption automatically means that a meconium test is negative, but even if this is so we feel it is still more than worth while to diagnose the remaining 85-90% at birth.—I am, etc.,

A. M. DEALL

Penrith,
Cumberland

"Stroke Units"

SIR,—In your leading article (6 November, p. 313) you suggest that we seriously consider the development of stroke units in this country, similar to those which have grown up in the United States. The conditions in the two countries are not, however, comparable. The majority of strokes occur in older patients and in the United Kingdom we already have a national network of geriatric units, which have done their best, with limited resources, to develop rehabilitation services. For many years these units have attracted stroke patients and many important contributions to the British literature of stroke have originated from such departments.

When the geriatric assessment unit of the Glasgow Royal Infirmary group of hospitals was established at Lightburn Hospital in 1968 one ward of 30 beds (15 male and 15 female) was set aside for stroke rehabilitation. A few patients are admitted from their own homes direct to this unit, but most strokes occurring in the area served by the hospital group are admitted in the first instance to the general medical wards of Glasgow Royal

Infirmary, where they undergo intensive care, if appropriate, and have ready access to neurosurgical and investigative facilities. Many patients are treated to conclusion in the medical wards, but each year between 150 and 200 patients are referred by the physicians in charge to the geriatric service, and are transferred, after assessment, to the stroke ward in the geriatric unit. One-quarter of these patients are under the age of 65, but none has objected to being treated alongside older patients. The stroke rehabilitation ward is organized along the lines of a standard geriatric rehabilitation ward, with close collaboration between doctors, nurses, physiotherapists, occupational therapists, speech therapist, medical social worker, and after-care services. The additional activities include a daily recreation programme organized by a ward hostess, pre-discharge home assessment, post-discharge follow-up, a club for relatives, group speech therapy, and group therapy for relatives of aphasic patients. A day hospital is being planned.

This arrangement has worked well, and similar collaboration between general physicians, neurologists, neurosurgeons, and geriatricians might prove a suitable pattern for the management of stroke patients in Britain. The unit depends heavily on its small staff of physiotherapists, occupational therapists, and speech therapists, and the continuing shortage of these grades of staff is the major obstacle to the needed expansion of stroke rehabilitation throughout the country.—I am, etc.,

BERNARD ISAACS

Department of Geriatric Medicine,
Lightburn Hospital,
Glasgow

Epitaph for the M.C.H.C.

SIR,—Dr. F. Nour-Eldin (30 October, p. 298) takes exception to the statement of Dr. M. S. Rose that neither the M.C.H.C. (mean corpuscular haemoglobin concentration) nor the M.C.H. (mean corpuscular haemoglobin) convey anything of particular clinical value any longer (16 October, p. 169) and then goes on to raise a number of points.

It must be made quite clear that Dr. Rose is referring specifically to the M.C.H.C. as calculated by automatic apparatus and is contrasting this with the M.C.H.C. derived from the haemoglobin level and the packed cell volume measured by manual methods. That these two figures do not always mean the same thing has now become obvious as is explained by Dr. J. M. England and others (23 October, p. 232).

(1) The M.C.H.C., as measured by the Coulter Model "S," does not fall in hypochromic anaemia except in the severest cases of iron deficiency. Therefore, when measured in such apparatus, it cannot "return to normal on successful treatment of hypochromic anaemias." The M.C.V. (mean corpuscular value), on the other hand, begins to fall relatively early in iron deficiency and is an excellent parameter both for assessing the degree of the deficiency and for following the response to therapy.

(2) The traditional teaching of textbooks of haematology is that the absolute values, the M.C.V. and M.C.H., are unacceptably inaccurate because they depend upon the red cell count, which has a large inherent error—of the order of $\pm 400,000$ cells out of a total of 5,000,000 cells if a visual count is done on 500-1,000 cells.¹ The introduction