baby to adopt. This is a fallacy: adoption societies exist to help babies and children offered for adoption to find a home, and they know from long experience that nothing less than the best is good enough for adopted children.

While mongolism may be least disturbing in a family, nevertheless to have a mentally handicapped child for an older sib is not the best environment for an adopted child, who will certainly have problems needing parental and family support in due course. Adoption societies are fully conscious of family psychodynamics, thinking particularly to teenage years, when there could be competition for parental time if there is a handicapped child of any description in the family. Case committees who make the decisions are very sensitive to these facts, and as they usually have more good homes without problems than babies available they are able to be selective. Furthermore, they think in terms of adoption families where two or more babies may be placed to give support to each other in time.

It would be a very fine couple to cope successfully with a mentally handicapped child and adopted children as a family. They would need resources and support within the family. I am sure that if a couple could present an adequate case that they had the requisite essentials they might get a sympathetic hearing from a reputable adoption society. But the society would need convincing, for no society would place a baby even in a home such as Dr. Bicknell recommends, where there is any possibility of that family remaining as two children.—I am, etc.,

JOHN PRICE.

Camberley, Surrey.

Fluctuating Course in Acoustic Neuroma

SIR,—The tendency for neoplasms to be progressive can hardly be challenged, but Dr. N. B. S. Sarkari and Dr. E. R. Bickstaff (5 April, p. 21) are to be congratulated for emphasizing the possibility of marked fluctuation of symptoms and signs before the progressive nature becomes apparent. They are correct in supposing that "an extrinsic tumour impinging on the brain stem might be found which might be capable of surgical eradication." Acoustic neuroma is an example, and remission is one feature (sudden onset is another) which can make diagnosis difficult.

In a series of 15 patients whom I have seen personally and who later had an acoustic neuroma removed eight showed some degree of fluctuation, and in seven this was sufficient to suggest an alternative diagnosis initially. In one a sensory deficit in the trigeminal area cleared up for several months, which seemed to confirm an earlier presumption of disseminated sclerosis, and in another recurring unilateral headache with visual disturbance suggested migraine. There was fluctuating hearing in two, acute episodes of vertigo in four, and recurrent facial palsy over a period of eight years in another.

This is something which needs to be stressed, since it is so much at variance with the usual textbook descriptions. Where doubts remain air or iophendylate in the ventricular system and vertebral angiography, as was suggested, will help, but may not

demonstrate a small tumour in the cerebello-pontine angle, which nevertheless would be outlined by myelencephalography.—I am, etc.,

Royal Infirmary,
Glasgow C.4.

J. A. DOIG.

Vitamin B₁₂, Serum Folate, and Hypochromic Anaemia

SIR,—The letter from Mr. P. H. Johnson and others (8 March, p. 643) prompts us to report similar findings, though our interpretation of these is different.

Of 60 patients with idiopathic iron deficiency anaemia 10% had a subnormal serum vitamin B₁₂, 27% a subnormal serum folate, and 38% an excess of hypersegmented polymorphs. Six of 42 had giant metamyelocytes in the marrow. The presence of abnormal myeloid cells was not always due to vitamin deficiency, for such cells were found when both serum vitamin B₁₂ and folic acid clearance, the most sensitive test of folate deficiency, were normal. This has been observed by others.^{1,2} This does not, however, exclude an altered metabolism of either vitamin in iron deficiency. The addition of folic acid to the iron therapy of those patients with a subnormal serum folate did not enhance haemoglobin response, nor was vitamin therapy needed to reduce the hypersegmented polymorphs to normal.

We agree, therefore, that vitamin deficiency is common, though in respect of folic acid the defect is slight, since no patient tested had a subnormal red cell folate. Low serum folates have been observed before in iron deficiency anaemia,^{3,4} but we do not consider that the vitamin deficiency is being masked by the iron deficiency, since iron therapy alone caused correction of the defect, in contrast to the exaggeration of the signs of vitamin deficiency observed when iron is given to patients with dimorphic anaemias.—We are, etc.,

D. W. DAWSON.
A. L. BUCKLEY.

Department of Pathology,
Crumpsall Hospital,
Manchester.

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Ophthalmic Disease and the Pill

SIR,—Ocular complications of oral contraceptives have aroused interest for some years. In 1965 Cogan¹ drew attention to certain neuro-ophthalmic affections being reported in the U.S.A., but doubted if they were related to the contraceptive pills. Walsh *et al.*² produced 69 case reports of neuro-ophthalmic interest, but the authors were not sure of the link between these diseases and the contraceptive pill.

A married woman aged 29 was admitted to the Royal Eye Medical Ophthalmological Unit on 3 October 1967 with a history that for four days she had noticed a black shadow in the lower part of the right visual field which had persisted. The patient had been taking megestrol acetate (Volidan) tablets for four years. Five months prior to this incident she had increased the dose to two tablets daily because of

breakthrough bleeding. On examination the right fundus showed an occlusion of the superotemporal arteriole with a corresponding field defect and a visual acuity of 6/6 part. Full investigation, including carotid angiography, revealed no abnormality. At present the right vision is 6/6 and the visual field defect is unchanged. The upper temporal arteriole is narrowed with sheathing and there is temporal pallor of the disc.

It seems clear that there is an association between taking oral contraceptives and some types of thromboembolic disease, perhaps due to an induced change in the clotting mechanism of the blood.³⁻⁵ The cerebrovascular system is probably involved in this correlation. With the patient reported there was no clinical or radiological evidence of an extracerebral source of the embolus to account for her retinal artery occlusion. We considered that megestrol was an important factor in this case. The relationship is impossible to prove and can only be suspected because of the rarity of this particular lesion in a young woman.

In addition to this case we have examined seven other patients with ocular disease, five with optic neuritis, one with central serous retinopathy, and one with superficial retinal haemorrhages, all of whom were taking oral contraceptives. We considered that, though the aetiology of these conditions is not fully understood, oral contraceptives should not be incriminated, as these conditions do occur in women during the reproductive years. It is difficult to evaluate cause and effect, as ocular side-effects of the pill appear to be rare.⁶⁻⁸ In Britain the Committee on Safety of Drugs has received few confirmed reports of ocular complications from these drugs.

Clinical trials with controls involving large numbers of patients would have to be carried out to assess the true incidence of ocular side-effects in view of the conclusion^{4,5} that thrombo-embolic disorders occur in 1 in 2,000 women on the pill.—We are, etc.,

JAMES C. McGRAND.
C. CHARLES CORY.

Royal Eye Hospital,
London S.E.1, and
King's College Hospital,
London S.E.5.

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Chronic Bacteriuria

SIR,—Your leading article "Chronic Bacteriuria" (15 March, p. 661) raises the question of bacteriuria in old age, its origin, and the desirability of treating it.

We have investigated the incidence of urinary infection in a normal elderly population—that is, all those aged 65 and over in a general practice in Beckenham—557 people.¹ In round terms we find the incidence of significant bacteriuria to be 20% in females over 65. In males over 70 the incidence was also

20%. Between 65 and 70, however, only 3% of men were affected. These figures agree closely with those in the only other published survey we know of the incidence of bacteriuria in non-institutionalized old people—that of Sourander in Finland.²

To discover at what age women acquire this high incidence of bacteriuria we are carrying out a survey of women aged 45 to 65 years in the same general practice. Preliminary findings show the following incidence of bacteriuria:

45-49 years	4% (of 118 women)
50-59 years	2.5% (of 164 women)
60-65 years	1% (of 80 women)

It seems likely that the high incidence of bacteriuria in old age has its beginnings in the late sixties in women and early seventies in men. We found a correlation (at 5% level) between bacteriuria and history of prostatic operation in men. There was, however, no correlation in women between bacteriuria and parity nor with a history of surgical interference at parturition nor with gynaecological operations.

We also treated with ampicillin a group of the women over 65 who were found to have infection.³ Of 22 patients who completed the treatment 10 had sterile urine three months later, but 12 remained infected or became re-infected. Symptoms of infection were higher in the former than the latter group, and most of these symptoms cleared after treatment. In those with persistent urinary infection there were few symptoms and these remained. Our findings therefore support your statement that many cases of bacteriuria in old age are chronic and refractory to treatment.

As to the cause of this high incidence of chronic bacteriuria in old age, we agree with your suggestion that obstructive uropathy is a common underlying factor. Since the incidence of infection in men and women is similar, it is unlikely that prostatic enlargement is the main cause of this. We believe that a common cause in old age is a functional obstructive uropathy associated with the neurogenic bladder. We have found evidence of a neurogenic bladder in a high proportion of non-incontinent elderly women⁴ and have suggested that the presence of uninhibited contractions, a bladder capacity of only 250 ml., and up to 100 ml. of residual urine are normal findings in a high proportion of elderly women. These changes are probably responsible for the high incidence of nocturnal frequency (70% of men and 64% of women over 65 get up at least once nightly to pass urine) and, we suggest, are also important factors contributing to the high incidence of bacteriuria in old age.—We are, etc.,

J. C. BROCKLEHURST.
J. FRY.

Lennard Hospital,
Bromley, Kent.

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Infection with Adult *Toxocara cati*

SIR,—A Sussex boy aged 4, who had been having frequent daily epigastric pains for four weeks, often connected with meals and lasting for half to one hour, vomited a specimen of *Toxocara cati* one morning after

drinking some warm milk. He was sick several times that day, but gradually over the next three weeks the pains diminished and finally disappeared. His stools were negative for worm eggs, as were those of his parents. He had no eosinophilia and in the differential count there was some shift to the left; the haemoglobin was 80%. A cat in the household was known to have worms and was probably the source of the boy's infection.

T. cati has been recorded as an adult infection of man about 18 times¹ from various countries, twice from England^{2,3} and twice from Ireland.^{4,5} It is normally and strictly a parasite of Felidae, among which there are 26 different kinds of hosts.⁶ Although *T. cati* is evidently a more frequent adult infection of man than its close relative *T. canis*, of which there appear to be only two cases on record,⁷ it is possible that some of the earlier reports may have been mis-identifications, since *T. cati* and *T. canis* can easily be confused. The specimen recovered in the present case is a male in fairly good condition and was identified with certainty as *T. cati*. It measures 36 mm. long by 1 mm. in maximum diameter; oesophagus 2.65 × 0.4 mm. with posterior ventriculus; cervical alae broad, 1.5 × 0.3 mm.; spicules, 1.9 mm. long; cuticular striations, 12–16 μ apart; tail 0.2 mm. long, typically probular; caudal papillae not well defined. It therefore represents only the third published occurrence of *T. cati* as an adult infection of man in England.

The apparent scarcity of *T. cati* in man in this country, where it is not uncommon in domestic cats, is notable and underlines the host specificity of the parasite. Its medical importance as an adult worm, unless many cases have been unreported, is therefore negligible. Its possible aetiology in the visceral larva migrans syndrome must be borne in mind, however, and worm-free cats as well as dogs should be a *sine qua non* in every household.—We are, etc.,

K. S. RODAN.

Department of Pathology,
Worthing Hospital,
Sussex.

J. J. C. BUCKLEY.

London School of Hygiene
and Tropical Medicine,
London W.C.1.

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Disodium Cromoglycate in Bronchial Asthma

SIR,—We are grateful to Dr. I. W. B. Grant (29 March, p. 842) for drawing our attention to the over-emphatic conclusion in the last paragraph of our paper (1 March, p. 552). His criteria for improvement are, however, no less arbitrary than our own, and he ignores the fact that only three of the eleven subjects had initial total scores for breathlessness, tightness, and wheeze which

were large enough to allow the fall of 126 points, regarded by him as being "modest but useful." A case in point was patient No. 4 whose symptoms were relatively mild while taking placebo (score 41 points) but who became asymptomatic (score 0) while taking disodium cromoglycate. This was surely more than the marginal improvement inferred by Dr. Grant. Any criterion used to assess whether a reduction in score is valuable must take into account the initial figure; thus halving of a small score after taking disodium cromoglycate may indicate an improvement which is well worth while to a patient.

The main object of our study was to find the physiological basis for the subjective benefit claimed by patients who take disodium cromoglycate when living south of the border. In the nine subjects who were subjectively better there was evidence of reduced airway resistance. This was best demonstrated by the use of the peak flow meter, because, we believe, this measurement was made twice daily. It is likely that the forced expired volume and the specific conductance of the airways would be equally reliable if performed with the same frequency, but this is scarcely possible for outpatients.—We are, etc.,

D. G. ROBERTSON.
D. A. WARRELL.

Department of Medicine,
Royal Postgraduate Medical School,
London W.12.

Anticoagulants after Myocardial Infarction

SIR,—It is truly regrettable that the Working Party on Anticoagulant Therapy in Coronary Thrombosis of the Medical Research Council (8 February, p. 335) devoted such great effort to determine the worth of anticoagulant therapy for the treatment of acute myocardial infarction only to find that by using the Thrombotest as a guide for dosage at levels of 10–20% they had administered the anticoagulant in less than recognized optimal therapeutic levels. As pointed out by Poller and Loeliger,¹ and as determined in our laboratory some years ago, a satisfactory level of therapeutic activity can only be maintained when the Thrombotest results are in the 5–10% range—7% is ideal. This range approximates the recognized therapeutic level of 15–23% by the Quick test.

The report of the Anticoagulant Committee of the American Heart Association (1954), emphasized the need for adequate therapeutic levels, noting that the rate of clinical thromboembolic complications occurring at dosage levels producing prothrombin activity of higher than 23% (Quick) was twice that occurring at levels of less than 23% activity. It was stated that "to receive full benefits of anticoagulant therapy, patients should be kept at all times at prothrombin times of at least 25 seconds (23% or less)."² For this study the Quick (Link-Shapiro) test was used with the control levels of 14–16 seconds. This concept has been widely accepted for all oral anticoagulant therapy, but was apparently not given sufficient consideration by those responsible for the protocols and administration of the studies of Hilden *et al.*,³ Seaman and Griswold,⁴ and the recent study of the M.R.C. Working Party. In each of these studies the results revealed a parallelism between the lack of adequate dosage and un-