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.,

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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.,

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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.,

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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-

satisfactory therapeutic response. The total available evidence suggests that had lower degrees of prothrombin activity been consistently maintained the results might have more closely resembled those of the Anticoagulant Committee of the American Heart Association,² Loeliger and his co-workers,⁵ and Meuwissen and Hart,⁶ Aspenström and Korsan-Bengtson,⁷ and the results of the U.S. Veterans Administration Long-Term Study,⁸ as well as many other studies both short- and long-term from the British Isles and from other countries.

The Working Party of the Medical Research Council had previously used an average daily dose of 108 mg. of phenindione in their study dealing with long-term preventive anticoagulant therapy, which produced favourable effects in several important categories of mortality and morbidity. However, in this study involving acute myocardial infarction the use of the Thrombotest at 10–20% levels resulted in a serious reduction of approximately one-third in the daily dose to an average of 72 mg. This represents a paradox in the principle of anticoagulant therapy. Acute thromboembolic states usually require more active rather than less active treatment when compared with long-term prevention in an otherwise quiescent phase. What this study does confirm is that when levels of activity in the range of 10–20% by the Thrombotest are used only minor therapeutic effects involving thromboembolic complications can be expected. As with all other potent drugs, one must give adequate amounts of anticoagulants to obtain full therapeutic effects.

It is a matter of great and international concern that clinicians not especially conversant with the problems and the research in this field will assume that the treatment was optimal, and thereby interpret this report as establishing that anticoagulants are ineffective in the treatment of acute myocardial infarction. This conclusion is not warranted—the therapeutic level used was not optimal.—I am, etc.,

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Avulsion of the Upper Limb

SIR,—Nearly 40 years ago I had a similar experience to that of Dr. Gunn, cited by Dr. Murray Drennan (29 March, p. 843). One Saturday in November I was driving home in the dark from shooting and came across

an accident. A woman of about 60, whom I knew well, had been knocked down by a large car travelling fast. The projecting door handle on the near side had caught her right arm and torn it clean off. There was a tag of mangled flesh which was bleeding in small pulsations and the woman was deeply shocked, but the ambulance had been sent for and arrived soon after I did, so that my contribution was merely to tie a piece of string tightly round the pedicle of the tag and send her off to the hospital.

For another twenty years she was a well-known figure in the village. An active and efficient housewife and a most cheerful and lovable character, she used to boast that her one arm could do better than a good number of younger pairs she knew.—I am, etc.,

GEOFFREY BARBER.

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SIR,—I have read with interest Dr. I. Barziano's three cases of upper arm avulsion (11 January, p. 118) and the following letters of Mr. J. A. Barclay (15 February, p. 444) and Dr. A. J. M. Drennan (29 March, p. 843). I have in my possession Cheselden's book *The Anatomy of the Human Body*, published in 1763, which describes the following case history facing an illustration.

"The figure of Samuel Wood, a miller, whose arm with the scapula was torn off from his body by a rope winding round it, the other end being fasten'd to the coggs of a mill. This happened in the year 1737. The vessels being thus stretched bled very little, the arteries and nerves were drawn out of the arm; the surgeon who was first called placed them within the wound, and dressed it superficially. The next day he was put under Mr. Ferne's care at St. Thomas' Hospital, but he did not remove the dressings for some days. The patient had no severe symptoms, and the wound was cur'd by superficial dressings only, the natural skin being left almost sufficient to cover it; which should in all cases be done as much as may be. Above twenty years since I introduced the method of amputating, by first dividing the skin and membrana adiposa, lower than the place where the operation was to be finish'd, the advantages of which are now sufficiently known."

—I am, etc.,

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Hypotensive Effect of Propranolol

SIR,—I read with much interest the paper by Dr. B. N. C. Prichard and Dr. P. M. S. Gillam on the treatment of hypertension with propranolol (4 January, p. 7) as well as a previous paper on the same subject (19 September 1964, p. 725).

I have observed a similar phenomenon in rabbits. Propranolol (5 mg./kg./day) administered to rabbits caused a significant drop in systolic blood pressure. The maximum hypotensive effect was observed 5–7 weeks after the onset of administration of propranolol, and hypotension was maintained for five weeks, at which time the experiment was terminated. I would like to suggest the rabbit as a good experimental model, as these results are very similar to those observed in man.—I am, etc.,

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Recovery after taking Weedol

SIR,—Dr. F. Kerr and his colleagues reported a case of Weedol ingestion with recovery (3 August, p. 290). I wish to give the details of three children who ate Weedol (which contains 5% paraquat) and recovered.

Three children, aged 3, 5, and 9 years, were admitted to hospital on 29 July 1966. They had been found by an older daughter. The 5-year-old was rolling around on the floor holding his abdomen and retching, but had not vomited. The youngest was just sitting very quietly (which was an unusual posture for him) and the oldest, at first quiet, began crying. A canister 4 in. by 1½ in. (10 cm. by 3 cm.) marked Weedol lay nearly empty on the floor, just a few granules being left in the bottom of the canister. No signs of granules scattered on the floor. On questioning, it transpired that another little boy who was present had brought the canister to the house and offered it to the children as icing decoration, and the three of them ate most of it between them. The eldest claimed that the youngest had most of it and she and the middle child much less. She said that the canister was full when they got it.

At that time the only reference readily available was Bullivant's report of the fatal outcome of two cases of paraquat ingestion,¹ but reference to the Edinburgh Poisons Bureau and I.C.I. Research Laboratories suggested that Weedol was not so lethal, there having been two patients who had eaten Weedol and recovered.

The patients were treated by gastric lavage, and by forcing oral fluids. This fluid intake was pushed so well by the nursing staff that all three patients were incontinent. All three patients progressed without complication, though the chest x-ray on the smallest child who was supposed to have eaten most of the Weedol was reported as showing "poor aeration at the lung bases." No attempt was made to analyse the urine for paraquat.

I wish to thank Dr. J. B. Borthwick, consultant physician, for permission to publish these cases.

—I am, etc.,

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Hypertension from Cold Remedies

SIR,—Commenting on hypertension in patients on monoamine-oxidase inhibitor drugs who take sympathomimetics—for example, in cold remedies—Dr. P. M. Humberstone (29 March, p. 846) writes "we do not have any evidence on how long to expect the blood pressure to remain raised in such circumstances."

In our experiments on normal volunteers taking monoamine-oxidase inhibitor drugs¹ we found rises in blood pressure from single oral doses of sympathomimetics lasted three to five hours. Naturally the height and duration will be influenced by dose, rate of absorption, etc., and may be longer and perhaps less with slow-release preparations.

Blackwell and Taylor² reported that 44% of 50 patients taking monoamine-oxidase