The second part of the same sentence states that "no one can yet offer a practical solution." You refer to the method advocated by myself and G. DeBlasio,¹ and conclude that "trials with the new method of assessment are not yet available." At the time of your writing you could not be aware of a trial of our method now in progress under the direction of Mr. J. Lannon of the Lady Dudley Hospital in Johannesburg, South Africa, for he has as yet not published his results of the past five years.

In a letter dated 2 July 1971 he states that as of that date he had used the method now in 749 operative patients all elderly with "not a single case of pulmonary embolism or leg vein thrombosis." He also states that he will not be without heparin prophylaxis in performing surgery. His experience parallels ours with the same success attained by us now, in 926 usually at risk patients. Before the use of heparin prophylaxis Mr. Lannon had indicated he had as we had experienced a 4% incidence of fatal thromboembolism in his operative patients.

I have no knowledge at this writing of others who presently use our prophylactic method and what their results are. I would also be happy to see a report of Mr. Lannon's experience but to-date he has not found the time to publish.

So perhaps you may find Mr. Lannon's assessment of our heparin prophylaxis as offering a "practical solution" to the prophylaxis of venous thrombosis for the operative patient at least.—I am, etc.,

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¹ Sharnoff, J. G., and DeBlasio, G., *Lancet*, 1970, 2, 1006.

Aplastic Anaemia and Leukaemia

SIR,—A case resembling those presented by Drs. I. W. Delamore and C. G. Geary (26 June, p. 743) has been reported by Gmyrek and others.¹ An 8-year-old girl with Fanconi type of aplastic anaemia received prednisone and an anabolic steroid and developed after a period of three years acute leukaemia of myelomonocytic type. Although Fanconi's anaemia might be potentially more leukaemic than aplastic anaemia of other type, there are only a few reports of this occurrence.—I am, etc.,

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Kotka,
Finland

¹ Gmyrek, D., Witkowski, R., Syllm-Rapoport, L., and Jacobasch, G., *Deutsche medizinische Wochenschrift* 1967, 92, 1701.

Gastric Ulcer and Gastritis

SIR,—Your leading article (10 July, p. 62) draws attention to the association of gastric ulcer and atrophic gastritis and cites evidence suggesting that the latter precedes the former. Whatever the chronological connexion between the two conditions, the observation is of interest for the pathogenesis of gastric ulcer because it is not long ago that both duodenal and gastric ulcers were classified as peptic ulcers. Hyperacidity whether of

nervous or hormonal origin was believed to play an important part in their pathogenesis.

It is timely to go further and to suggest that not only have duodenal ulcers and the primary gastric ulcer a different pathogenesis but a clear distinction should also be made between the primary (classical) gastric ulcer and those which occur in association with lesions in the pyloric antrum or the duodenum (pyloric channel syndrome). While in the former a normal or more often a low gastric acidity is found, the same does not apply to gastric ulcers in the pyloric channel syndrome.

There is a distinct difference in the age distribution between the types of gastric ulcer. Bateson¹ found that duodenal ulcer reaches a peak in patients between 30 and 60 years, concomitant gastric and duodenal ulcers between 40 and 60 years, and solitary gastric ulcer between 50 and 70 years. My own series² of 350 cases with primary chronic gastric ulcer shows likewise that these ulcers are predominantly seen in late middle and in old age. It is therefore difficult to ascertain the significance of atrophic gastritis or of pyloric incompetence in the pathogenesis of these ulcers, for both conditions are frequently manifestations of physiological involutionary processes.

An outstanding feature in chronic primary gastric ulcer is the high incidence of calcified atherosclerotic lesions in the central blood supply of the stomach. Though atherosclerotic lesions in the abdominal aorta are a common finding in advancing years, I found³ that while 65% of patients with the classical gastric ulcer show calcified lesions of various degrees in the abdominal aorta between the ages of 50 and 60 years, these lesions are seen in only 23% of control cases of the same age group. Moreover, a detailed radiological study of the distribution of the calcified atheroma in gastric ulcer cases often reveals their location not only in the proximal portion of the abdominal aorta but also in the coeliac axis.³ Thus the classical chronic gastric ulcer seems to be a complication of atherosclerosis like coronary, internal carotid, or renal artery disease, where arterial insufficiency and obstruction are less often caused by involvement of the small arteries immediately supplying the affected organ but rather by atheromatous plaques in the more central arterial supply.—I am, etc.,

A. ELKELES

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¹ Bateson, E. M., *British Journal of Radiology*, 1969, 42, 598.

² Elkeles, A., *American Journal of Roentgenology*, 1964, 91, 744.

³ Elkeles, A., *Journal of the American Geriatric Society*, 1970, 18, 450.

Reflux and Hernia

SIR,—In your leading article entitled "Reflux and Hernia" (24 July, p. 205) you draw attention to recent work by Cohen and Harris¹ and comment that some of their views will cause consternation among the traditionalists.

This may, I suppose, be true; but they will not, I am sure, cause consternation among many surgeons with experience of operating on cases of gastro-oesophageal reflux. The occurrence of reflux without herniation has, of course, been found by many observers to be not so very uncommon,

even if less common than Cohen and Harris's findings might seem to suggest. And after conventional repair operations with restoration of normal anatomical relationships, and excluding cases with hernia recurrence, reflux symptoms, and perhaps demonstrable reflux, persist in many cases. In my own experience this occurred in some 66% of such cases, and, though symptoms were often much improved, in over 20% they were little, if at all, improved.

You quote Cohen and Harris as challenging the rationale of surgical repair of hiatus hernia. Their findings certainly challenge the rationale of the conventional type of repair. But I regard them as providing support for those of us who for some time have held that restoration of normal anatomy may not be followed by restoration of normal antireflux function and who, because of disappointing results from conventional repair operations, have adopted one of the gastroplication procedures. I hold that these procedures work so well because they provide a completely new antireflux mechanism—a valve mechanism. It is with interest, therefore, that I note Cohen and Harris's finding that surrounding pressure does not increase sphincter strength. In the plication operation I carry out, in fact, the oesophago-gastric junction is not over-reduced, but fixed in its normal position, as with conventional repair operations, and the new antireflux plication arrangement is constructed beyond the cardia. To date, this appears to be completely effective in controlling reflux.

By carrying out this procedure on cadaveric stomachs, and testing them at the laboratory bench it can readily be demonstrated that the flow of fluid from the stomach into the oesophagus is prevented. No part can be played in this by sphincteric action or by intra-abdominal pressure. It would seem that it must be due to the production of a new antireflux mechanism—a valve mechanism.

The conclusion to be drawn would seem to be that when gastric reflux causes troublesome symptoms or oesophageal damage, whether associated with a hernia or not, what is required is the creation of a new antireflux mechanism. Fortunately this can be done.—I am, etc.,

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¹ Cohen, S., and Harris, L. D., *New England Journal of Medicine*, 1971, 284, 1053.

Pupillary Paralysis after Tranquillizer

SIR,—I would like to report on an unusual side effect apparently due to pimozide (Orap, Janssen Pharmaceuticals). The patient was a 24-year-old female, diagnosed as suffering from simple schizophrenia. She was treated with phenothiazines for three weeks (initially thioridazine, followed by trifluoperazine), without improvement in her clinical state. She was started on pimozide 6 mg in the morning, and after two weeks the dosage was changed to pimozide 4 mg in the morning, 2 mg at night. There was some improvement in her symptoms, and after three weeks the dosage was increased to pimozide 4 mg b.d. Six days later she complained of mild Parkinsonian tremor of the hand and leg and immobile facies. This responded to

benztropine 2 mg t.i.d., and she was discharged from inpatient treatment on this dosage.

She began attending a psychiatric rehabilitation day hospital shortly afterwards, and one month after starting pimozide 4 mg b.d. her schizophrenic symptomatology appeared to be under control, but she was complaining of poor visual acuity. On examination there was paralysis of the ciliary muscle of both eyes, with fixed dilated pupils, and paralysis of accommodation. She was examined by an ophthalmologist at this time and no other abnormality noticed to account for this. Pimozide was reduced to 2 mg daily as a maintenance dose, and orphenadrine 50 mg t.i.d. substituted for benztropine. Her vision and pupillary responses to light and accommodation gradually returned to normal over the following two weeks. It would appear, therefore, that as well as causing Parkinsonian side effects in a dosage of 8 mg daily, pimozide caused paralysis of accommodation, and interfered with normal pupillary reactions.

I would like to thank Dr. A. D. Forrest and Dr. J. D. Smythies for permission to report on this patient who was under their care, and Dr. J. Cullen, of the Royal Infirmary, Edinburgh, for confirming the ophthalmological findings.

—I am, etc.,

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"Stiff Man Syndrome" and Trauma

SIR,—The stiff man syndrome is now a well recognized clinical entity.¹ There has been frequent speculation on aetiology but to date there is no convincing evidence for any of the postulated mechanisms. We wish to report a case which appears to fulfil all the criteria of this disease and which is so closely related to trauma that this may well be implicated in its mechanism.

The patient was a 53-year-old man in good health until three years before admission when a large book-case toppled from a truck, struck his back, and rendered him briefly unconscious. For several months following the accident he had continuous pain in the paraspinal muscles, exacerbated by movement but never completely abating. He also developed increasing stiffness in his back and shoulders. The pain gradually subsided and disappeared over some months, but the stiffness was progressive until it involved the legs and arms. There was sudden exacerbation with emotional upset or sensory stimulus, often as little as a light touch on the skin, and this often resulted in excruciatingly painful cramping in the muscles of the abdomen and thighs. One year prior to admission he became unable to walk unassisted.

Physical examination revealed marked rigidity of all muscles of the trunk and legs. He was unable to bend from the waist. Gait was slow and unsteady, and he moved en bloc. The muscles were "bony" hard to palpation, and passive movement revealed continuous high tone in agonistic and antagonistic muscle groups. Stretch reflexes were brisk throughout with flexor plantars and no clonus. There was no muscle weakness or wasting. Muscle tone was, however, noted to be normal during sleep. Full evaluation by the psychiatric service revealed

no significant functional component to the man's illness.

Laboratory studies revealed a maturity onset diabetes, first demonstrated two years prior to admission. X-ray spine and myelography revealed only spinal straightening and mild degenerative disease. Nerve conduction studies were normal. Electromyography revealed continuous normal motor action potentials in the paraspinal muscles despite all attempts by the patient to relax. 10 mg diazepam intravenously led to electrical silence after 15 minutes, and this persisted for 60 minutes.

Treatment was started with 60 mg diazepam orally, with good result. The patient became able to walk unaided and showed only minimal residual paraspinal stiffness. Attempts to substitute a placebo and then diphenylhydantoin for diazepam were quite unsuccessful, the patient suffering severe relapse which on one occasion required catheterization for complete urinary retention which was believed due to muscle spasm. Favourable improvement has been maintained on diazepam for one year.

We present this as a probable case of stiff man syndrome after back trauma. Whether it is related in some way to the spinal damage or is secondary to months of continuous voluntary tension in the paraspinal muscles as a result of pain is not clear. Finally, hysteria cannot be entirely ruled out.—We are, etc.,

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University of North Carolina,
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¹ Gordon, E. R., Januzko, D. M., and Kaufman, L.,
American Journal of Medicine, 1967, 42, 582.

Treatment of Urinary Infections

SIR,—It was interesting to read that Dr. J. A. Davies and others (24 July, p. 215) in their comparative double blind trial of the treatment of urinary infections found that cephalixin and ampicillin were equally effective.

The logical conclusion, although not mentioned in their article, is that ampicillin must be preferred because of the enormous expense of cephalixin. In the dosage that they recommend the cost for five days treatment with ampicillin is £1.23 and with cephalixin it is £5.53. These prices are taken from *Mims* July 1971.

If a drug has some special indication cost is immaterial. If, however, two drugs perform identically, as in this case, the cheaper should be recommended. Is it not time that the medical profession became a little more cost-conscious about the treatment that it prescribes? I feel that this sort of information should be included in articles of this kind to help doctors to select the drugs that they use knowing all their advantages and disadvantages.—I am, etc.,

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Hong Kong Influenza Variant

SIR,—Dr. P. W. Gill (31 July, p. 308) points out certain features of recurrent epidemics of influenza A and suggests that infection

with the strains of influenza A₂ virus current in 1966 and 1968 may have protected against the Hong Kong variant which appeared in Australia in 1968 and in Britain in 1969. He asks that other doctors who have the appropriate records will publish them in order to provide evidence of the truth or otherwise of the suggestion.

The records from our general practice (3,600 population) show that during the dominance of the Asian types of influenza A₂ between 1957 and 1968 the Asian type virus was isolated from 118 persons. The Hong Kong variant was not isolated from any of these persons in the first Hong Kong influenza epidemic, but in the second epidemic it was isolated from five of them and from a person known to have had influenza A virus infection in the past. The dates of the five "Asian" type A₂ virus isolations were as follows: 1958 February, 1963 March, 1964 February (two isolates), and 1966 January.

Our experience therefore does not support the suggestion that the 1966 epidemic conferred protection against the Hong Kong variant. It does not contradict the suggestion that the 1968 Asian strains did so, because none of the 48 persons who were virus positive for the Asian type of influenza in 1968 developed Hong Kong influenza in either of the epidemics.

The fact that in the first Hong Kong epidemic all those persons known to have been previously infected with Asian type of virus escaped may be considered to support the hypothesis of a temporary protection against the Hong Kong variant. Did they escape fortuitously because it was such a small epidemic in which only some 5% of our population was attacked? Alternatively, was it a small epidemic because so many of our population had been temporarily protected by a previous attack of Asian virus?

I hope that others with accurate information will help to swell the evidence for or against the hypothesis proposed by Dr. Gill.—I am, etc.,

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Psychogeriatric Services

SIR,—The controversy which has been provoked by the report of the Society of Clinical Psychiatrists¹ suggests that the profession may be about to embark on a rather unedifying demarcation dispute.

Though patients can never rigidly be categorized, the problem of demented old people is best considered by dividing them into three broad groups.

(1) The bed-fast, who, owing to physical disabilities require heavy nursing care—for example, those crippled by strokes, Parkinsonism, arthritis, or contractures. These are currently, and rightly, a geriatric commitment.²

(2) The ambulant dement without any gross disturbance of behaviour. These must remain a community responsibility and ideally should stay in their own homes with increasing support from their relatives and social services. Thereafter they may live in ordinary local authority welfare homes, or in special residential homes for the mentally frail. In either event, their medical needs are