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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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.,

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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