choice of consultant is not important. But even in general medicine or surgery some G.P.s would put greater trust in the judgement of one consultant and others in that of another. In psychiatry this choice of consultants is still more important. Psychiatric diagnosis and treatment are very largely based on the relationship between the patient and the psychiatrist, perhaps more exclusively so than in other medical specialties. Moreover, there are numerous schools of psychiatric thought in existence which differ greatly in their diagnostic and therapeutic outlook and terminology. Some of these schools are even irreconcilable with each other. G.P.s either may themselves subscribe to one of those schools of thought and perhaps abhor others or may think that a particular psychiatric outlook may be more useful in a specific case than another. I needed advice from a psychiatrist with psychotherapeutic experience and knowledge which Dr. Z had not. The institution of catchment areas left me with a "choice" of exactly one psychiatrist, who was unsuitable in this particular case. This seems to me a ludicrous, dangerous, and—from the point of view of a free profession—degrading situation.

I understand from the local medical committee that the consultant psychiatrists have intimated that catchment areas enable them to provide a more efficient service. I cannot possibly comprehend what "a more efficient service" could mean in this context, and heaven help us if efficiency in this sense merely means that more patients can be put through some kind of psychiatric mincing machine. Apparently it is not only the bureaucrats who are taking our freedom away but also our own colleagues. Are there G.P.s left who are prepared to fight for professional freedom and the choice of consultants that it implies?—I am, etc.,

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Fibrinolysis and Venous Thrombosis

SIR,—Your leading article "Fibrinolysis and Venous Thrombosis" (16 November, p. 365) is inaccurate in several respects. Ethyloestrenol is not an oestrogen but an anabolic steroid of progestogen type with androgenic properties. The proprietary name is Orabolin.

Allenby *et al.*¹ did not use phenformin plus ethyloestrenol but ethyloestrenol alone, and their operations were performed at a time when, in our experience, resistance to the fibrinolytic effect of ethyloestrenol given without phenformin would be expected to be developing.² Furthermore, Professor I. M. Nilsson's work should not be associated with the use of these drugs to prevent post-operative venous thrombosis. Her patients were in a steady fibrinolytic state whereas postoperative patients show a considerable fibrinolytic "shut-down,"³ which the drugs may well not be powerful enough to combat. Your leading article is therefore likely to confuse those not working in this field.—I am, etc.,

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³ Chakrabarti, R., Hocking, E. D., and Fearnley, G. R., *Journal of Clinical Pathology*, 1969, 22, 659.

It is, of course, possible that the causes of postoperative deep vein thrombosis and recurrent idiopathic venous thrombosis are not the same. Nevertheless, the depressed fibrinolytic activity seen after surgery is, in some respects, similar to the reduced lytic activity demonstrated by Professor Nilsson and her colleagues in patients with recurrent idiopathic venous thrombosis, though further research is needed to confirm this similarity.

Neither of the clinical trials quoted in our leading article induced sufficient stimulation of fibrinolysis in surgical patients. As Dr. Fearnley states, one trial used only one drug and the other gave both drugs for too short a time. We pointed out that further trials were therefore needed to show whether phenformin and ethyloestrenol given in the correct dose for long enough could prevent both postoperative venous thrombosis and the decreased fibrinolytic activity that follows surgery.—Ed., *B.M.J.*

Lightning

SIR,—Your leading article (26 October, p. 181) reminded me how short memory in medical literature appears to be. In 1946 I was commissioned by the Editor of the *Lancet* to write a leading article on lightning injuries.¹ The main reason for my being asked to do so was my close co-operation at the time with the late Professor Stephan Jellinek, who was the pioneer of research concerning electrical and lightning injuries. His classic *Elektrische Verletzungen* is still the standard textbook of electropathology.² Apart from Vienna, he worked for many years at Oxford and it was there that I got to know him well. Other people in Britain who studied such effects on the central nervous system were Macdonald Critchley³ and Duncan Leys.⁴ It seems a pity that the references of your leading article appear to suggest that the greater part of present knowledge of the pathology and clinical features of lightning injuries dates from the 1960s and the beginning of this decade and that the 50 years of thorough and fruitful research carried out by Jellinek—for nearly 20 years in Britain—is forgotten.—I am, etc.,

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¹ *Lancet*, 1946, 1, 351.

² Jellinek, S., *Elektrische Verletzungen*. Leipzig, Barth, 1932.

³ Critchley, M., *Lancet*, 1934, 1, 68.

⁴ Leys, O., *Edinburgh Medical Journal*, 1942, 49, 657.

Ascorbic Acid Deficiency and Aspirin-induced Haematemesis

SIR,—Your leading article, "Aspirin and What Else" (6 July, p. 5), and subsequent correspondence discussed the mechanisms by which aspirin induces haematemesis. Professor H. C. Stewart (24 August, p. 525) referred to the possibility that subclinical scurvy could be responsible for haematemesis after aspirin. Plasma and leucocyte ascorbic acid concentrations progressively diminish during life, reaching minimal values at the

age of 60.¹ This age-induced physiological tissue desaturation could certainly predispose to the haematemesis associated with aspirin consumption in older patients. Pathophysiologically-induced ascorbic acid desaturation of ascorbic acid occurs with many diseases.² Many drugs induce tissue desaturation of ascorbic acid, including nicotine from cigarettes, alcohol, several appetite suppressants, iron, phenytoin, some convulsant drugs, the oestrogen component of oral contraceptives, and tetracycline.^{3,4} Aspirin is one of the most powerful drugs producing ascorbic acid tissue desaturation in normal individuals.⁵

Pathophysiological and drug-induced ascorbic acid tissue desaturation occur in gastric and duodenal ulceration,^{6,7} after gastric surgery,⁸ and in association with gastrointestinal haemorrhage.⁹ Symptoms of subclinical scurvy appear.¹⁰ Haemorrhage results from the ascorbic acid desaturation associated with a 45% reduction in platelets and a 50% increase in prothrombin time.¹¹ Ascorbic acid loss from platelets is directly related to their degree of aggregation. The lost ascorbic acid is not released into the plasma but is probably utilized for metabolic purposes during aggregation.¹² After aspirin is given to human subjects adrenaline-induced platelet aggregation is minimal and ascorbic acid is not lost. Barnes and his colleagues¹³ showed that adrenaline-induced platelet aggregation is abnormal in patients taking as little as 150 mg of aspirin. This dose can produce haemostatic ineffectiveness.

You inquire why an occasional person has profuse gastric haemorrhage after taking aspirin. Ascorbic acid tissue levels and metabolic utilization vary greatly between individuals under normal circumstances. During disease, reduction in plasma ascorbic acid concentration due to metabolic demand can profoundly alter platelet activity, diminish vascular stability, and increase bleeding and clotting times. Ascorbic acid tissue desaturation would exacerbate any existing platelet abnormality. Administration of aspirin in these circumstances would not only produce tissue desaturation of ascorbic acid but also inhibit its normal metabolic utilization by the tissues and thus contribute to the bleeding tendency.—We are, etc.,

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¹ Loh, H. S., Wilson, C. W. M., *International Journal for Vitamin and Nutrition Research*, 1971, 41, 258.

² Wilson, C. W. M., *Vitamin C: Tissue Metabolism, Oversaturation, Desaturation and Compensation in Vitamin C*, ed. G. G. Birch and K. J. Parker, p. 203. London, Applied Science Publishers Ltd., 1974.

³ Wilson, C. W. M., *Proceedings of the Nutrition Society*. In press.

⁴ Wilson, C. W. M., *Annals of the New York Academy of Sciences*. In press.

⁵ Loh, H. S., Watters, K., and Wilson, C. W. M., *Journal of Clinical Pharmacology*, 1974, 13, 480.

⁶ Cohen, M. M., and Duncan, A. M., *British Medical Journal*, 1967, 4, 516.

⁷ Dymock, I. W., *et al.*, *British Medical Journal*, 1967, 2, 375.

⁸ Williamson, J. M., Goldberg, A., and Moore, F. M. L., *British Medical Journal*, 1967, 2, 23.

⁹ Russell, R. I. *et al.*, *Lancet*, 1968, 2, 603.

¹⁰ Wilson, C. W. M., *Practitioner*, 1974, 212, 481.

¹¹ McCraw, J. Y., *Review canadienne de biologie*, 1956, 14, 295.

¹² Sahud, M. A., and Aggeler, P. M., *Proceedings of the Society for Experimental Biology and Medicine*, 1970, 134, 13.

¹³ Barnes, R. M., Evans, P. R., and Smith, D. S., *British Medical Journal*, 1974, 3, 412.

¹ Allenby, F., Jeyasingh, K., and Calnan, J., *Lancet*, 1973, 2, 38.

² Fearnley, G. R., and Chakrabarti, R., *Journal of Clinical Pathology*, 1964, 17, 328.