of the written language in any other way. Such patients may also be unable to conceptualise the meaning of a two-dimensional health educational film and fail to internalise the message. Efforts to bridge the social, cultural, and dietary gap to explain a complex disease is as daunting a task as the problem of communication itself. The use of competent interpreters to communicate with such Asian patients is the most effective and practical answer to this problem. I am glad to read that Dudley Road Hospital has found this to produce worthwhile results.

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Perspectives in spina bifida

SIR,—I wonder if I might raise a rather academic point in connection with a matter mentioned in your leading article on spina bifida (30 September, p 909).

In the third paragraph it is said that "body weight increases as the cube of the surface area, power only as the square." I think this is wrong, but I would be interested in knowing your comments. So far as I remember body weight would increase as the cube of the linear dimension and power as the square of the linear dimension. As the surface area is a function to the power of 2 of the linear dimension, I think for this reason the quoted statement is incorrect. At any rate I remember being told in connection with the bridges the Romans built that their models stood upright whereas the actual bridges collapsed and that the arithmetic was as I have said.

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*Mr Redman is probably correct in suggesting that the phrase should be "body weight increases in proportion to the cube of the surface area" rather than "as the cube of the surface area." His mathematical intervention emphasises the problem even more strongly. It was interesting to learn that little Roman bridges stayed up whereas big Roman bridges fell down, and this is most certainly true of what happens to little patients with spina bifida when they grow up into big patients with spina bifida.—ED, *BMJ*.

Effects of drugs on myocardial necrosis after subarachnoid haemorrhage

SIR,—In their article concerning the protective effect of propranolol and phentolamine on myocardial necrosis following subarachnoid haemorrhage (7 October, p 990), Mr G Neil-Dwyer and his colleagues assign a negligible protective effect to phentolamine on the grounds of "probable absence of α -receptors in the myocardium." There is, in fact, increasing evidence for the presence of such receptors,¹ but even if they are of little significance compared with myocardial β -receptors one cannot dismiss the role of α -receptors in the coronary vasculature in the production of ischaemic change.

Alpha-receptor-mediated coronary vasoconstriction has been clearly demonstrated in animals,² and in man the use of phentolamine

in the treatment of vasospastic angina has abolished angiographically demonstrated coronary artery spasm.³ In addition to its cardiovascular effects phentolamine has an inhibitory effect on central sympathetic outflow⁴ and stimulates insulin secretion,⁵ which may have a protective effect on myocardial metabolism.

Thus, although we would agree that on the basis of the animal studies quoted by the authors it is likely that β -blockade was the main factor in protecting against myocardial necrosis, we feel it is unjustified to dismiss the role of phentolamine until a study using propranolol alone has been performed.

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¹ Rabinowitz, B, *et al*, *American Journal of Physiology*, 1975, **229**, 582.

² Schwartz, P J, and Stone, H L, *Circulation Research*, 1977, **42**, 79.

³ Levene, D L, and Freeman, M R, *Journal of the American Medical Association*, 1976, **236**, 1018.

⁴ Schmitt, H, and Schmitt, H, *European Journal of Pharmacology*, 1970, **9**, 7.

⁵ Taylor, S H, in *Phentolamine in Heart Failure and Other Cardiac Disorders*. Berne, Huber, 1975.

Polymyalgia rheumatica and primary biliary cirrhosis

SIR,—Abnormalities of the standard liver function tests, particularly raised alkaline phosphatase levels, are well recognised in polymyalgia rheumatica (PMR). However, such abnormalities do not confirm the diagnosis of primary biliary cirrhosis (PBC) and we do not feel that the three cases reported by Dr J C Robertson and his colleagues (21 October, p 1128) have been adequately proved to have this disease in association with PMR. Only one patient (case 2) had a liver biopsy, and the pathological findings suggest cirrhosis superimposed on a chronic hepatitis. A case of PMR with similar liver histopathology has recently been described.¹ The other two patients did not have liver biopsies and therefore it is impossible to prove the diagnosis of PBC.

Although up to 94% of patients with PBC have positive antimitochondrial antibodies (AMA),² the converse is not true. Positive AMA have been described in systemic lupus erythematosus and the collagenoses, in which the incidence is 8% in contrast to 0.8% in the general population.³ A specific systemic lupus erythematosus-like syndrome with positive AMA was described in Germany in 1973, and subsequently in your columns.^{4 5} This syndrome consists of myalgia, arthralgia, fever and a raised erythrocyte sedimentation rate with the association of pleurisy and pericarditis; 30% of the patients have a raised plasma IgM level.⁶ We would suggest that it is possible that two of the patients (cases 1 and 3) described by Dr Robertson and his colleagues have variants of this syndrome.

With regard to the previously reported case of PMR with PBC cited by Dr Robertson and his colleagues, Walker *et al*⁷ stated categorically that they considered this patient to have had an aggressive chronic hepatitis and that since there was no evidence of bile duct necrosis on liver biopsy a diagnosis of PBC could not be supported.

We therefore feel that an association between PMR and PBC has not yet been proved. We

also feel strongly that a diagnosis of PBC cannot be made without a liver biopsy.

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¹ Thompson, K, and Roberts, P F, *Postgraduate Medical Journal*, 1976, **52**, 236.

² Doniach, D, Walker, J G, and Roitt, I M, *Acta Gastroenterologica Belgica*, 1968, **31**, 399.

³ Berg, P A, Doniach, D, and Roitt, I M, *Journal of Experimental Medicine*, 1967, **126**, 277.

⁴ Guardia, J, *et al*, *British Medical Journal*, 1975, **1**, 370.

⁵ Florin-Christensen, A, *et al*, *British Medical Journal*, 1975, **1**, 513.

⁶ Doniach, D, and Walker, J G, *Gut*, 1974, **15**, 664.

⁷ Walker, J G, Doniach, D, and Doniach, I, *Quarterly Journal of Medicine*, 1970, **153**, 31.

Treatment of rheumatoid arthritis

SIR,—Dr J M Gumpel describes his personal views (14 October, p 1068) on the management of rheumatoid arthritis (RA). We accept many of his opinions, but the use of gold only in patients with erosions or progressive joint space loss needs a comment.

It has been shown that gold treatment is able to retard the development of erosions in patients with RA.^{1 2} As early as 1935 Forestier³ stated that gold is effective in particular in the early stages of RA. We have shown gold treatment to retard most effectively the advance of erosions at the early stage of RA, 10 months or less from the onset of the disease.⁴ If gold treatment was initiated later the result was worse.

The main purpose of gold treatment is to prevent the destruction of joints. Already existing erosions cannot be prevented. If gold treatment is initiated late, at the erosive stage of the disease, the patient cannot become symptomless any more. In our opinion gold treatment ought to be started in the early stages of RA, before the development of erosions. Some patients with benign disease may receive gold unnecessarily, but the benefits of prevention of disability in some patients are so great that this risk is tolerable. It must be seen that we are treating not only the actual inflammation of the joints but also the quality of the patient's life during many decades in the future.

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¹ Sigler, J W, *et al*, *Annals of Internal Medicine*, 1974, **80**, 21.

² Luukkainen, R, *et al*, *Scandinavian Journal of Rheumatology*, 1977, **6**, 123.

³ Forestier, J, *Journal of Laboratory and Clinical Medicine*, 1935, **20**, 827.

⁴ Luukkainen, R, *et al*, *Scandinavian Journal of Rheumatology*, 1977, **6**, 189.

Acute reaction to apple-eating

SIR,—Adverse effects of apple-eating have been previously reported^{1 2} and Havland and Frithz described the case of a woman who developed Stokes-Adams attacks after apple ingestion.³

A 27-year-old doctor consulted a senior colleague because of distressing symptoms which were induced by apple ingestion. These attacks had started five years previously and consisted of sweating, faintness, and extreme nausea, which would come on about 15 seconds after swallowing an apple. He had never lost consciousness during the attacks, which were always brought on by