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.

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

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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

apple-eating and seldom lasted for more than one minute. No other food provoked these symptoms, which also occurred when the peel was removed or eaten separately and when only a small piece of apple was eaten. Cooked apples produced no reaction whatsoever. The symptoms could be reduced slightly by drinking a glass of cold water immediately after swallowing the apple. Physical examination was completely normal. His brother, who was also a doctor, gave a very similar history, but in his case the symptoms were much less severe. Both of them also suffered from hay fever and had learned to avoid apples altogether.

Because of a possible cardiac effect³ electrocardiography was performed during apple ingestion. Both the resting trace and the trace during swallowing and the following 3 minutes showed no abnormality and the pulse and blood pressure (erect and supine) remained normal throughout despite the presence of his usual symptoms.

The aetiology of this patient's symptoms remains obscure. The rapidity with which the symptoms develop after ingestion, their brief duration, their alleviation by drinking water, and the absence of cardiac effects suggest that a local mechanical or chemical effect of the apple pieces on the oesophagus may be responsible. It is interesting that both he and his brother have a history of atopy, and the lack of effect of cooked apples perhaps indicates the role of a heat-labile antigen.

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¹ Mackarness, G R G, *British Medical Journal*, 1973, **1**, 114.

² Johnstone, I L, *British Medical Journal*, 1972, **4**, 613.

³ Havland, T, and Frithz, G, *British Medical Journal*, 1976, **2**, 643.

Educational needs of handicapped children

SIR,—Never before have the problems and needs of the physically handicapped been publicised so much as they are now, from the highest governmental echelons to the grass roots of local voluntary bodies. You are therefore to be commended on following this desirable trend in your issue of 4 November by publishing the touching article by Margaret Blackwood (p 1292) and your leading article on the educational needs of handicapped children (p 1245). Your editorial concludes with a pointed and poignant reference to current financial stringencies impeding the recommendations arrived at after lengthy deliberations by experts.

The problem of educating physically handicapped children becomes the concern of doctors because many of these children are on drugs—anticonvulsants, sedatives, etc—or have disabilities—the incontinence of spina bifida children or the ataxia of the cerebral palsied. Many of these unfortunate children have severe multiple handicaps and require individual assessment and attention by their teacher. I feel there is insufficient liaison between the prescribing doctor and the teacher responsible. I do not believe that this individual care can be given when attempts are made to integrate the children into ordinary schools or even in the few specialised schools that exist for the physically handicapped. As you point out, research studies have indicated that a high teacher:pupil ratio is required—sometimes as high as 1:2. How can the country afford all this?

I think that an answer can be found by

setting up small tutorial units for these children in groups of about six. Special schools would not be required since local authority clinics, which are already structurally adapted for mothers with prams or for wheelchairs and are mostly built on one level, could be used. Nearly all the clinics I know are not fully utilised during the day. There is no shortage of teachers or voluntary helpers to assist these children not only in conventional school subjects but also in occupational therapy, speech therapy, simple games, etc.

Health centres owned and run by group practices could play an important and even pioneering role here. It could even be a useful augmentation of the practice income if rental could be obtained from the local authority or even from parents if the organisation can be run on private lines. I would here draw attention to the fact that local authorities have a statutory obligation to provide for the education of the physically handicapped child and I have known certain education authorities pay up to £600 per term when they cannot provide proper facilities for this themselves.

If we are to help the physically handicapped to play a useful and independent role in society and not become permanent charges on public funds for the rest of their lives it is imperative to start early and this means providing a proper and tailored education for these children, who so often display a courage and cheerfulness that put our moans and grumbles to shame.

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Link between hepatoma and hepatitis B

SIR,—We would like to correct an error made in your leading article on this subject (9 September, p 718).

The statement is made there that "in a recent retrospective pathological study of hepatoma from California fewer than 1% of non-cirrhotic patients with hepatoma had markers of hepatitis B virus infection, whereas 73% of patients with cirrhosis had HBs antigenaemia." Reference was made to a paper from our liver unit.¹ The 73% figure was clearly stated in our paper to be the frequency of identification of HBsAg in the serum of patients with hepatocellular carcinoma (HCC) arising in *non-alcoholic* but cirrhotic livers. The decision to separate the patients with HCC in non-alcoholic cirrhosis from those with HCC in alcoholic cirrhosis seemed reasonable when, after immunoperoxidase staining of the liver of 168 patients with HCC dying between 1949 and 1970, an inverse relationship was found between racial groups with a high occurrence of HCC in alcoholic cirrhosis (Caucasians and Mexicans) and racial groups with a high frequency of occurrence of HCC in B-viral cirrhosis (Orientals and Negroes). Thus since 1969, when serum studies became available, alcoholic cirrhotics with HCC have been studied separately from patients with HCC in B-viral cirrhosis.^{2,3}

It is of less importance that the editorial also errs when quoting our incidence of HBsAg with HCC in normal livers to be less than 1% instead of the 5% stated in our paper.

At our centre the most common liver disease underlying HCC is still alcoholic cirrhosis. However, when comparing the coincidence of HCC and B-viral disease with that of countries in which alcoholic cirrhosis is nearly non-existent it seems only

appropriate to use only HCC in *non-alcoholic* cirrhosis as the denominator.

In your leading article you further state that "such a difference has not been found in major series from Britain and the United States." You then give reference to one study of frequency of HBsAg in HCC in Vietnamese⁴ and to a second article "in press." We are unaware of any other large study in which comparison of HBsAg in HCC patients from the United States, Britain, or Western Europe was made with HCC patients from Asian and African countries after exclusion of the alcoholic cirrhotic. Since our patients were predominantly from the indigent population, study of a different socioeconomic group might result in a different coincidence. The table would indicate that the racial differences in relationship of HCC to HBsAg are reduced after exclusion of the alcoholic. It is likely that the two racial groups with the highest alcoholic rates (Mexican and Caucasian) still include some alcoholic cirrhotic patients who steadfastly denied alcoholism and whose cirrhosis was not identifiable as alcoholic type.

Frequency of HBsAg positivity in hepatocellular carcinoma arising in non-alcoholic chronic liver disease in adults, California, USA

Race	No of patients	Positive for HBsAg (%)
Oriental ..	19	89
Negro ..	15	80
Caucasian ..	14	78
Mexican ..	14	57
Total	62	77

The fact that the frequency of finding HBs-antigenaemia in patients with HCC arising in *non-alcoholic* cirrhosis in the USA is similar to that in the Orient and in Africa suggests that if there is a cocarcinogenic factor it must be one common to or of similar potency in widely disparate geographical, agricultural, and cultural regions. It seems more likely that differences in frequency of occurrence of hepatocellular carcinoma in various geographical areas is related to differences in the incidence of chronic B-viral disease and perhaps the time in life at which hepatitis B virus was acquired.

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¹ Peters, R L, Afroudakis, A P, and Tatter, D, *American Journal of Clinical Pathology*, 1977, **68**, 1.

² Peters, R L, in *Hepatocellular Carcinoma*, ed K Okuda and R L Peters, ch 8. New York, Wiley, 1976.

³ Omata, M, et al, *Gastroenterology*. In press.

⁴ Welsh, J D, et al, *Gastroenterology*, 1976, **70**, 392.

Failure of mecillinam and pivmecillinam to eradicate convalescent carriage of salmonellas

SIR,—The apparent efficacy of pivmecillinam¹ in curtailing chronic salmonella carriage^{1,2} has prompted us to assess the usefulness of this drug and that of mecillinam in a group of convalescent faecal excretors of non-typhoid salmonellas. We gave both drugs by mouth.

Eighteen patients aged between 21 and 95 years (mean 69 years) received a total of 22 14-day courses of treatment under inpatient supervision. Known duration of excretion