Correspondence

Correspondents are asked to be brief.

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Recurrent Meningitis

SIR,—We have read with interest the medical memorandum on recurrent meningitis by Dr. O. G. Brooke and others (24 October, p. 218). From the accompanying x-ray films we would think that the incidental cystic findings at myelography are indeed sacral nerve root cysts, originally described in 1938 by Tarlov and then called perineurial cysts.¹ Although the association in this case suggests a possible cause for the meningitis, we feel that the two are probably not related since the track of the sinus leads not to the cysts but rather to the most caudal part of the subarachnoid space.

Sacral nerve root cysts are an occasional myelographic finding and are most likely to be shown when the patient is rescreened after an interval. This necessity for delayed screening may be a factor accounting for the infrequent reports of sacral cysts. We believe that sometimes the cysts may be the

cause of sciatica in a patient who is diagnosed as having a disc protrusion and in whom it is subsequently not possible to confirm this either by myelography or at operation.

During the past three years we have encountered eight such patients, who have recently been reported elsewhere.² In three of our patients the cysts were an incidental finding, while in the other five they were felt to be causing symptoms. Four of these latter patients were operated on in order to obliterate the cysts and relieve symptoms.—We are, etc.,

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- 1 Tarlov, I. M., *Archives of Neurology and Psychiatry*, 1938, 40, 1067.
- 2 Plewes, J. L., and Jacobson, I., *Lancet*, 1970, 2, 799.

Tennis Elbow

SIR,—Two years ago metal tennis racquets became widely available. From my own experience and that of others I believe that they have a marked tendency to cause tendonitis at vulnerable sites. Classical tennis elbow refers to tendonitis of the extensor aponeurosis at its insertion into the lateral epicondyle, but other common conditions are medial epicondylitis and bicipital tendonitis.

I had played tennis for 18 years without ever having had trouble with my arm, until I played regularly with a "Smasher"—a racquet made of aluminium alloy and manufactured by Spalding Company. During my first year of using this racquet I developed severe bicipital tendonitis which subsided only after total abstinence from the game during the winter months. After three months of play this year I developed medial epicondylitis which later extended to include both sides of the elbow. Then a friend told me that he had also had pain, first in the shoulder, and later in the elbow, a few

months after he commenced using a "Smasher." But he said that his arm had returned to normal when he recommenced using a wooden racquet. The connexion struck me as fortuitous until the day I took out my wooden racquet for the first time in two years. I instantly noticed that the exquisite pain previously experienced at the moment of contact with the ball had gone. The impact had a diffuse quality which was in contrast to the sharp jerk from the metal one. From that moment I used a wooden racquet, and my elbow improved.

The advantage of the "Smasher" is that it is light and at the same time rigid so that the player can hit the ball very hard, but in return the ball hits his arm very hard. The shock wave passes unimpeded up the metal, whereas wood, with its natural suppleness, absorbs much of the impact. Presumably the sudden stretch, transmitted by the metal racquet, exceeds the elastic limit of the tendons.

I then spoke to the third man (not Orson

Welles) in this tale. Although he had played a lot of tennis and won many trophies while using wooden racquets, he had never had any trouble with his arm. However, after using a metal racquet regularly, he developed increasingly severe forearm pain which was maximal over the medial epicondyle. After returning to the use of a wooden racquet he experienced progressive relief.—I am, etc.,

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Peripheral Gangrene after Measles

SIR,—We read with interest the case report of gangrene of digits after chickenpox by Drs. O. H. B. Gyde and D. L. Beales (31 October, p. 284). This prompts us to report a case of peripheral gangrene in a child following measles due to presumed intravascular coagulation.

A boy aged 2 years was admitted on 10 June 1969 with bronchopneumonia six days after the onset of measles. On admission he was acutely ill, pyrexial (104°F.; 40°C.), dyspnoeic, and cyanosed. There was clinical and radiological evidence of bilateral bronchopneumonia. Repeated sputum culture and throat swab did not grow any pathogenic organisms. As he did not respond to ampicillin and cloxacillin, and his white count was only 2,300/cu.mm., with lymphocytes 54% and atypical mononuclear cells 5%, the possibility of viral pneumonia was considered. The presence of a few groups of multinucleated giant cells in the sputum raised the possibility of giant cell pneumonia which is a rare complication of measles. Twenty-four hours after admission he became drowsy and comatose, and he had several bouts of generalized convulsive seizures. He had bilateral extensor plantar responses with a normal C.S.F. The clinical picture was compatible with measles encephalitis. The subsequent course was stormy, and to complicate matters further nine days after admission he developed several small

purpuric eruptions on the trunk and limbs. On the following day he developed bluish discoloration of several toes of both feet suggesting early gangrene. The skin overlying the area was warm and the peripheral pulses were palpable. The purpuric eruptions became more extensive and he had several bouts of haematemesis. At this stage, the possibility of disseminated intravascular coagulation was considered. No specific diagnostic laboratory test was then available. However, his platelet count was only 25,000 cu.mm. and thrombocytopenia is probably a constant feature in intravascular coagulation.

It was decided to treat the child with continuous intravenous heparin. The decision to anticoagulate the child was not an easy one as we were then faced with the paradoxical situation of gangrene on one hand and spontaneous bleeding on the other. After 48 hours of heparin therapy the child's condition improved remarkably, with rise in platelet count and cessation of spontaneous bleeding. The progress of gangrene was halted. During the course of several days the discoloration of the toes gradually disappeared, apart from small black necrotic areas at the tip of two toes. Heparin therapy was discontinued after seven days. Apart from loss of nail beds of his toes his feet are now perfectly normal.

The diagnosis of intravascular coagulation in this case was made mainly on clinical grounds and it does not fulfil the criteria of this syndrome as suggested by Corrigan and others¹—namely, the combination of the thrombocytopenia, reduction of coagulation factors II, V, VIII and fibrinogen, and presence of fibrinolytic split products in the serum, but the dramatic response to heparin therapy was gratifying and possibly diagnostic. Disseminated intravascular coagulation or consumption coagulopathy, a relatively new terminology, is well documented in relation to virus diseases.²—We are, etc.,

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Temporary Cardiac Pacing Using the Subclavian Vein

SIR,—We were delighted to see the paper by Drs. M. B. Macaulay and J. S. Wright (24 October, p. 207). We hope that these authors' experience with supraclavicular percutaneous introduction of the pacing electrode into the subclavian vein will popularize this little-known technique. Wider appreciation of its enormous advantages over arm-vein routes in terms of electrode stability and comfort for the patient should do much to counter the nihilistic attitude towards the treatment of postinfarction heart block which now prevails in too many centres.

We adopted the subclavian technique some three years ago and can confirm its simplicity, safety, and patient-acceptability. Thanks to clear instructions from Dr. B. W. Lassers and the enthusiasm of Dr. J. H. N. Bett (now of Melbourne, Australia) the

method proved easy to learn. The left subclavian vein is preferred here where temporary pacing is required in patients with chronic heart block so that later use of the right internal jugular vein (for insertion of a permanent electrode) is not prejudiced. Use of the left subclavian may also be more convenient for the right-handed operator and the natural curve of the temporary electrode favours correct placement of the tip in the right ventricular apex when introduced from the left side. There are unimportant differences in the equipment used and one potentially important difference—that is, we use only No. 5 F.G. bipolar electrodes, having known the stiffer No. 6 F.G. electrodes to perforate the heart in times gone by.

The Table shows the few complications we have encountered in 60 attempts to establish pacing with the subclavian technique and compares them with the

TABLE.—Comparison of Subclavian and Antecubital routes for Temporary Pacing

	Subclavian Vein	Antecubital Vein
Technique Attempted ..	60	19
Pacing Established ..	54	16
Failed Procedure* ..	6	3
Subsequent Electrode		
Displacement ..	2	8
Phlebitis ..	0	2
Wound Infection ..	0	2
Patient Discomfort ..	±	+++

(*includes 2 subclavian artery punctures due to inexperience)

complications of 19 temporary pacing procedures attempted during the same period using antecubital veins. No instances of pneumothorax, brachial plexus injury, or air embolism have been encountered. The latter complication might be anticipated in patients with low central venous pressure¹ unless tilted head down, and in those with obstructive airways disease and uncontrollable coughing; the subclavian technique is unsuitable for use in such cases.

Failure to introduce the electrode into the subclavian vein is now rare (a foam plastic pad, 8 cm. thick, beneath the occiput appears important to success). In a recent case where difficulty was encountered, the patient suffering recurrent asystolic arrest throughout, satisfactory pacing was speedily established via the right internal jugular vein as described by Hoffman and Sokol.²—We are, etc.,

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Malaria in Britain

SIR,—There are still several deaths every year¹ in Britain from malaria. A man aged 55 was recently admitted who had been in Nigeria for two weeks, but was not offered or advised about prophylaxis for malaria. Ten days after his return he developed symptoms of a chest infection, and was treated with antibiotics. Five days later he collapsed and was admitted to hospital in coma and profound shock. Cerebral malaria was considered, and the blood film showed heavy infection with *Plasmodium falciparum*. Despite intensive treatment his condition slowly deteriorated, and he died a week later.

At necropsy there was very extensive cerebral involvement with numerous haemorrhagic foci having necrotic centres, surrounded by deposits of malaria pigment. In retrospect it is unlikely that he had any chance of survival after the onset of coma.

There is still, despite pressure from many quarters, no one body responsible for providing advice about malaria prophylaxis for travellers. The responsibility must surely lie with both general practitioners and travel agents to provide this much needed advice. This previously fit man was the fifth death due to malaria this year in England—he had no prophylaxis, and the diagnosis was not made until too late. In this age of air travel, it is vitally important to think of tropical disease in anyone who has recently returned from an endemic area.

Anyone who would like further information concerning malarial endemic areas, and recommended prophylactic schedules, should contact Professor P. C. C. Garnham, at the Malaria Reference Laboratory, Horton Hospital, Epsom, Surrey.—I am, etc.,

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¹ Department of Health and Social Security. *On the State of the Public Health. Annual Report for the Year 1968*, p. 36. London, H.M.S.O., 1969.

Skin Disease and the Gut

SIR,—I have read with interest your leading article (1 August, p. 240) and the subsequent correspondence (29 August p. 521, and 5 September, p. 586) on skin disease and the gut and also the latest contribution of Dr. Janet Marks and Professor S. Shuster on dermatogenic enteropathy (12 September, p. 618). I am writing to draw attention to the similarities between dermatogenic enteropathy and the enteropathy associated with extraintestinal malignant tumors—which can be termed as “neoplastic enteropathy”—and to suggest that folic acid deficiency is mostly responsible for the small bowel dysfunction in both conditions.

Although jejunal mucosal abnormalities have been described in the past in patients with extraintestinal neoplasia^{1,2} two recent investigations have shown that such changes are indeed very rare.^{3,4} However, as in dermatogenic enteropathy, malabsorption does exist in the absence of morphological mucosal abnormalities. While the incidence and severity of malabsorption is proportional to the extent of rash in patients with skin disease, they appear to be related to the presence and extent of metastases in patients with carcinoma.⁴

Folic acid deficiency is quite common in patients with extensive skin disease,⁵ as well as in patients with malignant neoplasia,^{2,4,6} and it is generally believed to be due to an increased demand for the vitamin by the rapid turnover of cells in the skin and the tumor respectively. Folate coenzymes play a key role in cellular division through their action in nucleoprotein synthesis and an impaired synthesis of D.N.A. is a major defect in folate deficiency.⁷ As the cell turnover in the intestinal epithelium is higher than in any other in vivo cell population,⁸ interference with normal regeneration of villi, with resulting malabsorption, is only to