

be of great importance for the prognosis of the tumour and in part may serve as guidelines for the mode of therapy to be chosen. Several other recent studies confirm this suggestion.²⁻⁵ Radical excision of the tumour and post-operative radiotherapy seem to be the choice of treatment when the degree of malignancy of the tumour is low (grade I). When the tumour is classified as highly malignant (grade II or III), the probability of later metastasis seems so great that therapy with a combination of cytostatic agents should be applied after surgery and radiotherapy.⁶ The advantages of limb amputation seems to be limited to only a few selected cases.

Until recently angiography has been little used in the diagnosis of soft-tissue sarcomas, since the evidence on the diagnostic advantages of this method has been to some extent contradictory. During the past decade the use of vasoactive agents has much improved the reliability of this method.^{7,8} A recent angiographic study on 35 soft-tissue sarcoma patients in the University Central Hospital, Helsinki, Finland, suggests that a preoperative angiography is very useful in defining the size and vascular supply of the tumour.⁹ Further, this study reports on the usefulness of pharmacangiography in defining the character as well as the size of the tumour. Tumour recurrences seemed, however, to present a radiological diagnostic problem.

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² Pinedo, H M, *Cancer Treatment Reviews*, 1977, **4**, 67.

³ Suit, H D, and Russell, W O, *Cancer*, 1977, **39**, 830.

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⁵ Russell, W, *et al*, *Cancer*, 1977, **40**, 1562.

⁶ Gröhn, P, *et al*, *Duodecim*, in press.

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The grumbling appendix

SIR,—While a scar in the right iliac fossa may be a comfort to Mr J W Maltby (1 September, p 555) when faced with an acute abdomen, it may also be a trap for the unwary. Firstly, the appendix may not have been removed if an alternative procedure was performed through an "appendix incision." Secondly, failure to remove all of the appendix leaves an appendiceal stump which may subsequently be the site of inflammation and even perforation, as the following case illustrates.

A 44-year-old woman from Japan presented with a 10-day history of increasingly severe right iliac fossa pain associated with vomiting and later with diarrhoea. She gave a firm history of appendicectomy for "severe appendicitis" performed in Japan 10 years previously. On the second and seventh days of her illness she consulted two separate general practitioners, both of whom diagnosed gastroenteritis. Examination revealed an ill, dehydrated lady with signs of peritonitis; there was a "gridiron" scar in the right iliac fossa. At laparotomy, performed after resuscitation, there was purulent free fluid and a large abscess cavity was found in the right iliac fossa between the caecum

and adjacent loops of small bowel; at the base of the abscess cavity was an acutely inflamed, perforated, appendiceal stump 1 cm in length. A further appendicectomy was performed together with peritoneal toilet. The patient made an uneventful recovery.

Although the management of the appendix stump has received much attention little emphasis has been placed on the need to excise the whole appendix. "Subtotal" appendicectomy results from failure to appreciate the full length of the appendix, because either it is kinked and bound to the caecum by adhesions or oedema of the adjacent caecum obscures the appendix base.

In the reported case it is likely that the history of appendicitis and the presence of the appropriate scar resulted in delay of diagnosis and treatment of what is still a lethal disease.

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Susceptibility to primary biliary cirrhosis

SIR,—We have read with great interest the article by Drs J G Douglas and N D C Finlayson concerning individual susceptibility and environmental factors for the development of primary biliary cirrhosis (18 August, p 419). In the two reported families, the authors did not find any association between HLA antigens and the disease, but they did not look for the HLA-DR antigens.

Recently we have studied 21 patients with primary biliary cirrhosis and we found an increase in DRW 3 (57.1%) in relation to the control group (14.8%) ($P < 0.004$). Details of this investigation are to be published in *Tissue Antigens*.

Since the antigen DRW 3 is mostly associated with autoimmune disorders, and it seems also to be related with primary biliary cirrhosis, the hypothesis of a genetic basis is strengthened. The presence of DRW 3 is not an absolute requirement, and some still unknown environmental factors may act as a triggers for the development of the disease, as suggested by Douglas and Finlayson.

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Serum bilirubin and hepatic enzyme induction

SIR,—I was interested to read the paper by Dr A K Scott and his colleagues (4 August, p 310). Their results confirm our report of low total bilirubin levels in epileptic patients receiving treatment.¹ I am less happy, however, than they are about the simplicity of measuring bilirubin levels in blood. Even with modifications to the basic Malloy and Evelyn method, such as that described by Michaelsson *et al*,² it is doubtful whether low total bilirubin concentrations can be reliably measured; this is especially so when one uses diazo methods to measure concentrations in the normal range. Not only the timing of venesection but also the

marked effects of feeding are well known. I would therefore caution against hoping that bilirubin levels can be used as an indicator of hepatic enzyme induction, attractive as it also seemed to us a few years ago.

I also wonder how a liver biopsy can be used to "assess hepatic enzyme induction."

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¹ Thompson, R P H, *et al*, *Lancet*, **1**, 21.

² Michaelsson, M, *et al*, *Pediatrics*, 1965, **35**, 925.

Spontaneous recovery from rapidly progressive glomerulonephritis

SIR,—The title chosen by Dr Douglas R Maxwell and others (15 September, p 643) appears somewhat unfortunate, since the "spontaneous" recovery followed prolonged dialysis. Moreover, the cases described had about 50% glomerular crescents, and although the term "rapidly progressive glomerulonephritis" is sometimes applied when the proportion of crescents is even smaller¹ to define a prognostic group it should probably be confined to patients with 70% or more crescents.² Even in such cases, and in the presence of oliguria, we have found the occasional patient who recovers useful renal function.³

A less misleading title might have been "Residual renal function in extracapillary glomerulonephritis treated by dialysis alone."

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¹ Olsen, S, *Acta Pathologica et Microbiologica Scandinavica* [A], 1974, **82**, suppl, p 249.

² Cameron, J S, and Ogg, C S, in *Glomerulonephritis: Morphology, Natural History and Treatment*, ed P Kincaid-Smith, T H Mathew, and E L Becker, part II, p 735. Yew York, John Wiley and Sons, 1973.

³ Richards, P, Evans, D J, and Wrong, O, *British Medical Journal*, 1968, **2**, 259.

Kidney transplants and long-term immunosuppression

SIR,—The paper by Dr F Di Padova and others (18 August, p 421) questions the necessity of long-term immunosuppressive therapy after kidney transplantation. We would like to report on a patient whose self-initiated withdrawal of azathioprine and steroid medication did not have such a favourable outcome for the function of his transplant.

The patient, a 20-year-old man, was first seen in August 1972, when chronic glomerulonephritis was diagnosed. In December 1972 chronic hemodialysis therapy was instituted because of end-stage renal failure, and in March 1973 he received a kidney transplant from a cadaveric donor. HLA-typing revealed poor histocompatibility, with one mismatch at the A and two mismatches at the B locus. Immediately after the transplantation a good graft function was obtained, and except for one acute rejection episode in the early post-transplant period no further complications were observed. Immunosuppressive therapy consisted of azathioprine and prednisolone as usual. Under a regimen of 100 mg azathioprine and 7.5 mg prednisolone a day the patient had an excellent transplant function for five years, his plasma creatinine concentration being consistently about 133 $\mu\text{mol/l}$ (1.5 mg/100 ml).