inclined to this view. Perhaps Dr Newton and his colleagues can reanalyse their data excluding all those life events (some of which were listed by Dr Roland) that are open to subjective interpretation.

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[2] Brown, G W, *et al*, *Psychological Medicine*, 1973, **3**, 74.
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Samaritans versus suicide

SIR,—Minerva asks (22 September, p 743) "Just how effective are the Samaritans in preventing suicide?"—a question impossible to answer owing to lack of controls. Many of us have personal experience of individual cases where suicide has definitely been averted by our intervention; but these cases cannot be enumerated.

The following data, however, were extracted from the records of a random sample of 57 224 of the 250 740 individuals who sought our help during 1978. A total of 13 421 callers admitted suicidal thoughts or intentions, and 15 763 callers admitted no such thoughts or intentions; in 28 040 cases for various reasons this information was not available. Thus 45·9% of the 29 184 cases in which the topic of suicide was broached (either by the caller or by the Samaritan volunteer) admitted to suicidal ideas; or, to put it another way, 23% of the whole sample were found to be harbouring them. This gives the population a "potential" suicide rate of 23 453 per 100 000, whereas the official figure is only about eight per 100 000. Evidently we do attract those with a suicidal bent, but quite a lot of them think better of it.

Minerva also states that the suicide rate is showing no further decrease despite our increased activity. True. It is levelling out at about eight per 100 000, having dropped steadily from 12 per 100 000 in 1963 (coincidentally the year when Samaritans Incorporated came into action). One of the possible reasons for its not falling further may be that the number of our new callers each year has increased since 1973 by 60% whereas the number of our volunteers has risen only 17%. Many of our 170 branches are overstretched; to achieve some sort of parity with this increasing work load we need at least 6000 more volunteers—urgently.

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High- and low-carbohydrate diets

SIR,—Dr K W Heaton of Bristol Royal Infirmary has written to us pointing out that the dietary fibre figures given in our paper concerning the use of high and low carbohydrate diets in maturity onset diabetes (30 June, p 1753) differ from those calculated using the new McCance and Widdowson food tables.[1] The discrepancy has arisen for two reasons.

Firstly, at the time the calculations were made the new tables were not available and we used preliminary figures given in the *Journal of Human Nutrition* (Vol 30, Nos 5, October 1976, p 303), which for some foods differed quite considerably from the definitive version. Secondly, our calculations included the fibre content in the skin of soft fruit. The correct figures for the high- and low-carbohydrate diets should be 64 g and 17 g respectively. Clearly the overall conclusions of the study are not affected in any way by this discrepancy.

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Cancer of the ovary

SIR,—We were interested to read your leading article (22 September, p 687) on ovarian cancer but question the therapeutic conclusions, which exclude radiotherapy from consideration.

"Advanced" ovarian cancer is an ill-defined term. Any discussion of treatment must take into consideration the known routes of spread to the para-aortic lymph nodes and diaphragm. The latter should be examined in all cases as metastases may otherwise be missed.[1] Internal mammary node involvement may be detected by lymphoscintigraphy and is probably of importance in the pathogenesis of ascites.[2] The role of cytoreductive surgery is now well established and it has been found that it is the amount of residual disease that affects prognosis rather than initial extent of the disease.[3]

What therapies should be used as adjuncts to surgery? For many years radiotherapy was the only treatment available. Its role has now been reinvestigated in a prospective randomised trial at the Princess Margaret Hospital, Toronto.[4] Some stage I cases probably need no treatment other than surgery if staged adequately with lymphangiography and laparoscopic examination of the diaphragm. A prospective study to test this hypothesis is in progress at the Royal Marsden Hospital; patients are followed closely with repeated laparoscopy to detect recurrence early should it occur.

In stages II and III whole-abdominal radiotherapy has been shown to be effective in eliminating microscopic disease in patients with no visible residual tumour[4]; unfortunately few patients fall into such a favourable group. For those with bulky residual disease throughout the abdomen after maximal surgical resection chemotherapy is appropriate. Such "advanced" cases do indeed require determined treatment with both modalities.

The techniques of radiotherapy need further evaluation. Pelvic irradiation alone is often inadequate because of the high frequency of abdominal relapse.[4] The whole abdomen may be irradiated either by opposing fields or by a moving strip technique. Supplementary dosage may be directed to the pelvis, para-aortic lymph nodes, and diaphragmatic lymphatics.[5] The instillation of intraperitoneal colloidal radioactive isotope may have a promising future.[6] It is of theoretical interest that its absorption follows the route of spread of cells released into the peritoneal cavity.

Future progress will depend on well-organised prospective randomised clinical trials with internationally agreed criteria of response. Chemotherapy is at an early stage of its development; although response rates of 50-60% are a marked improvement, they have yet to be translated into long-term survival. This is most likely to be achieved by interdisciplinary co-operation in the management of these patients.

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[4] Dembo, A J, *et al*, *Cancer Treatment Reports*, 1979, **63**, 249.
[5] Glatstein, E, Fuks, Z, and Bagshaw, M A, *International Journal of Radiation Oncology, Biology and Physics*, 1977, **2**, 357.
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SIR,—I consider your leading article on ovarian carcinoma (22 September, p 687) to be a fair review of the epidemiology of the disease. However, I believe every statement in the final two paragraphs is open to question.

The recommendation that at laparotomy for inoperable disease as much tumour as safely possible should be removed may seem reasonable—although no operations are entirely safe, and I would query the use of radical but macroscopic surgical techniques in treating a disease which by definition metastasises and invades initially at a microscopic level. I am not clear how a maximum response to therapy of a disease with such a varied natural history can be assessed—so when (if at all) should a repeat laparotomy be undertaken? If further "adequate" chemotherapy is required to eradicate residual tumour, is this to be established by a third laparotomy, and how long should this cycle continue? Personally (and not surprisingly) I have found in-vitro techniques for assessing tumour response to chemotherapy in vivo to be disappointing.

I deplore the use of survival figures in the final paragraph as a sole measure of prognosis. Enthusiasm is an important motivation to those involved in attempting to overcome such a common and formidable disease. However, I would urge them to remember that clinicians must treat their patients as individuals and not as survival figures, and that there may be occasions when relapse and rapid death may be a merciful release not only from the illness but from radical surgical expertise and determined chemotherapy.

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Soft-tissue sarcomas

SIR,—Your recent leading article (8 September, p 562) emphasises correctly the therapeutic problems in treating soft-tissue sarcomas. The guidelines in soft-tissue sarcoma treatment are highly variable, probably owing to the fact that many of the reports on soft-tissue sarcoma investigation are based on a relatively small number of patients.

A recent study on 153 soft-tissue sarcoma patients,[1] treated in the University Central Hospital, Helsinki, Finland, suggests that both the histopathological diagnosis and especially the grading of the tumour seem to

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 pharmacoangiography 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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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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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 smaller[1] 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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[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.
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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 μmol/l (1·5 mg/100 ml).