between populations of "alcoholics" and "normal drinkers." Moreover, Ledermann's hypothesis has not only been disputed, but is generally recognised to be incorrect,[1 2] so that the text here is also misleading.

The third diagram is a graph of three variables, all inadequately described, displayed on a single vertical scale. While the consumption and spending variables have units of measurements associated with them, albeit of an incomplete nature, the third—relative cost—does not. Why should relative cost start from a value of 7 in 1960? We might be led to conclude from this graph that in 1970 the relative cost of alcohol was equal to the per capita consumption while the percentage spending on alcohol was rather higher. Such a comparison of incommensurables is nonsensical. So is the graph.

The pictogram carries no indication of what is represented by one man. While the picture does carry the legend "SMRs for selected occupations," it is left to the reader to infer that these are for cirrhosis mortality. The interlocking circles in the next diagram are unsatisfactorily labelled. Who are to be considered problem drinkers? All binge drinkers or only a small proportion of them?

Lastly, not too much significance should be attached to the entries in the final two tables. There is considerable dispute over the validity of the methods currently used to obtain estimates of prevalence of, and costs associated with, excessive alcohol consumption.

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[1] Skog OJ. *Br J Addict* 1980;**75**:133-45.
[2] Duffy JC. *Br J Addict* 1980;**75**:147-51.

******Dr Paton writes: "Jennifer Waterton has missed the point of the various 'ABC' series, which are meant to depict concepts visually with the minimum of text. One of the directions to authors is that so far as possible the illustrations should not have legends. They are in no way to be considered as mathematical or statistical representations."—ED, *BMJ*.

Rectal examination and acid phosphatase

SIR,—Mr P Shridhar and others (15 August, p 502) report in your columns elevation of immunoreactive acid phosphatase at 30 minutes after rectal examination in patients with "prostatic and non-prostatic disease."

At present the standard tests in most laboratories for acid phosphatase are those using various substrates for the enzyme, rather than measurement of immunoreactive mass by a radioimmunoassay. We appreciate that immunoreactivity does not necessarily correlate with biological activity. However, it would have been very valuable if Mr Shridhar and his colleagues had carried out a concomitant assay for enzyme activity on the same samples used in the radioimmunoassay as an internal control for their study. If they did indeed get rises of the magnitude they reported, these would very likely have been detectable by measuring enzyme activity. Since our results and those of many others quoted in our paper (25 April, p 1378) showed no such rise, the only explanation would be that all the immunoreactive mass liberated during the rectal examination is totally lacking in enzyme activity; and this is quite unlikely.

Their report is rather devoid of details. For example, it would be important to know which patients did and which did not show a rise. The age range of their patients was from 12 to 86 years; the younger patients, if they were prepubertal, would not have much acid phosphatase in their prostates and if they showed a rise this is likely to be due to factors other than the rectal examination per se. Their reported increase was up to 14-fold. It is difficult to imagine how a 30-second rectal examination, presumably without even massage of the prostate, can result in a 14-fold increase in the serum pool of the enzyme.

We await publication of their detailed results with interest, hoping to have answers to questions such as how the baseline levels were established, how much variation there was in levels with time, how the samples were treated, and whether the assays of samples taken before and after the rectal examination were carried out together, and a discussion of whether there could be an alternative explanation for the observed rise.

In the meantime, we would maintain and reiterate our conclusion that, so far as acid phosphatase levels measured by enzymatic activity are concerned, it is a myth that there is a rise following rectal examination.

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******This letter has unfortunately been much delayed because the original copy never reached us, and also because we tried to get a reply from Mr Shridhar, who is now abroad. —ED, *BMJ*.

Hepatitis B infection in glomerulonephritis

SIR,—Dr H Rashid and others (10 October, p 948) suggest that hepatitis B virus infection is a rare cause of glomerulonephritis in Britain in contrast with the findings abroad. This would be consistent with the fact that there have been very few British reports of this association despite the routine screening for hepatitis B surface antigen (HBsAg) performed in all cases of chronic glomerulonephritis considered for haemodialysis. I can report similar negative findings in a series of cases from the West of Scotland.

Forty-six patients with glomerulonephritis (32 children, three with membranous glomerulonephritis; 14 adults, four with membranous glomerulonephritis) were investigated. HBsAg was sought in renal biopsy material from all patients by the immunoperoxidase method of Turbitt.[1] No evidence of HBsAg was found. In addition, review of the patients' notes failed to reveal any evidence of current or previous hepatitis.

Polyarteritis nodosa has also been linked with hepatitis B virus infection.[2] Review of the literature on this association also reveals a paucity of cases reported from British hospitals. In the postmortem records of Glasgow Royal Infirmary I found 30 cases of histologically proved polyarteritis nodosa in which sections of liver were available. Review of the liver histology in these cases showed no evidence of active or previous hepatitis. Staining of the liver sections for HBsAg by the same immunoperoxidase method produced uniformly negative results, suggesting that there was no carriage of the virus.

These results agree with Dr Rashid's conclusion that hepatitis B virus is not a major cause of glomerulonephritis in Great Britain and also suggest that it is not a major aetiological factor in polyarteritis nodosa in this country.

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[1] Turbitt ML, Patrick RS, Goudie RB, Buchanan WM. *J Clin Path* 1977;**30**:1124-8.
[2] Trepo CG, Zuckerman AJ, Bird RC, Prince AM. *J Clin Path* 1974;**27**:863-8.

Spirochaetosis: a remediable cause of diarrhoea and rectal bleeding?

SIR,—We read with interest the two case reports by Drs J G Douglas and V Crucioli (21 November, p 1362) associating intestinal spirochaetosis with diarrhoea and rectal bleeding. We have seen a 31-year-old woman who complained of intermittent diarrhoea, abdominal discomfort, and distension of three years' duration, a history that has been described in association with intestinal spirochaetosis.[1] Sigmoidoscopy revealed a mild proctitis and spirochaetes were demonstrated in the brush border of a rectal biopsy specimen stained by haematoxylin and eosin and in electron micrographs. The diarrhoea settled without treatment and spirochaetes were not demonstrated in further specimens, but abdominal discomfort and distension persisted and were not relieved by a course of oral metronidazole.

Using a technique developed for the isolation of *Treponema hyodysenteriae*, the agent of swine dysentery, we have isolated spirochaetes from this patient and from rectal swabs of passive male homosexuals with no intestinal symptoms.[2] The spirochaetes differed, particularly in that the organisms from the patient described could be cultured only with difficulty and could not be subcultured. This suggests that different spirochaetes may be associated with the human bowel; they may be pathogens, or alternatively may be commensals which proliferate on an abnormal intestinal mucosa. We suggest that the pathogenicity of this group of organisms remains unproved and further study is required.

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[1] Gad A, Willen R, Furugard K, Fors B, Hradsky M. *Upsala J Med Sci* 1977;**82**:49-54.
[2] Tompkins DS, Waugh MA, Cooke EM. *J Clin Path* (in press).

Facial burns due to fan heater

SIR,—The report by Drs F Van Genachten and G Tudor-Williams (14 November, p 1299) made me wonder just how safe these heaters were.

It was in order to be able to answer this sort