the patients with an important service that is now not available, and by reason of his 18 months of urology he would be able to recognise more difficult conditions of the urinary tract, where referral of the patient to a specialist urological unit would be in the patient's best interest.

If we are going to be able to continue to provide a service to the community in the surgical specialties, such bridging of sub-divisions of surgery will be necessary. The closed-shop attitude of surgical specialties is to be deplored. I think that surgeons in training who wish to practise general surgery should spend between one and two years obtaining special expertise in other branches of surgery such as urology, vascular surgery, gastroenterology, and endoscopy, so that they can offer a higher standard of care in these subspecialties and still do their share of general surgical work. Such a simple and straightforward solution to unsatisfactory contemporary practice would require a change of attitude in those who espouse the doctrine of closed guilds.

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SIR,—Your leading article "Second-best prostatectomy?" (1 March, p 590) is the second such article in the *BMJ* in the last two years. On each occasion the case for the specialist urologist as an endoscopic prostatectomist has been advanced. Of course, in an ideal world all patients with prostatism or acute retention should be referred to a specialist urologist, as should patients with urinary tract infections, suspected ureteric colic, etc. Waiting lists for treatment would soon increase to such an extent that elective surgery would probably come to a standstill and treatment as an emergency become the order of the day. It is bad enough to wait six years for hip surgery but would it not be intolerable and often dangerous to wait such a time for prostatic surgery? I trust we are not returning to the era of the coiled catheter secreted within a black top hat?

Through miscalculation, or more probably the unfulfilled promise of finance for new urological posts, the urologists find an excess of highly trained young men with nowhere to go. There are many senior registrars in surgery who have an interest in urology but who do not wish to lose contact with the practicalities of general surgery. They could surely be more readily accommodated at a district general hospital than a specialist who feels uncomfortable about being on call for general emergencies yet is prepared to perform parathyroidectomies and anastomose the small bowel as a routine.

To say that the general surgeon with an interest in urology does not exist is as true as saying the urologist with an interest in general surgery does not exist. As your article points out, 80% of prostatectomies in England and Wales are performed by general surgeons. If the general surgeon with an interest in urology does not exist who, pray, is performing surgery on these patients?

If you had read my article "Prostatectomy at a district general hospital?"¹ you would have noted that the overall mortality was 14 in 600 (2.3%), 12 of the patients dying from cardio-respiratory disease—but in this group there was no significant difference in mortality between open operations and transurethral

resections. In case you select the 54-year-old man who died with mitral valve disease, actually he had a transurethral resection. Below the age of 70 (75% of patients) the mortality was 1%, from 70 to 80 years 2.2%, and over 80 (13% of patients) 7.4%.

There is a place for the specialist urological surgeon but it would be better for him to accept that the general surgeon with an interest in urology (who after all seems to do much of the work) does exist and can and should be taught to provide a safe and adequate service without relinquishing his interest in general surgery.

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¹ Leach RD. *Ann R Coll Surg Engl* 1979;61:459-62.

SIR,—Having just done a transurethral prostatectomy in which I resected 100 g of prostate tissue I was somewhat disturbed to read in your leading article (1 March, p 590) on prostatectomy that I do not exist. I was under the impression that I was a general surgeon with a special interest in urology.

Perhaps in an ideal world all urology would be done by urologists but we have to face the economic facts of life and I believe that the urological service provided by the general surgeon who devotes most of his time to urology is better than your article suggests. One will remember that the great names in urology in the recent past, in fact the founders of British urology, were general surgeons who later developed their interest in this specialty. A good urologist must be a good general surgeon capable of the surgery of the intestine, major blood vessels, adrenals, parathyroids, etc; while the general surgeon by building up the necessary experience can become a useful urologist, needing to refer only a small proportion of his work to a special unit.

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Ingestion of household cleaning products

SIR,—For the period January 1974 to June 1975 calls about household cleaning agents received by the London centre of the National Poisons Information Service were retrospectively surveyed.¹ Of a total of 1457 cases, only a small proportion was followed-up in detail. Even so, the data were judged sufficient, along with other relevant published reports, to conclude that: (a) the intrinsic toxicity of most household cleaning products in the UK was not high; (b) there may be a risk of exaggerating the toxic hazards which household cleaning products present to the young; and (c) excessive medical treatment for cases of this kind should be abjured.

To check whether this state of affairs still prevailed three years later, another survey was carried out. This time it covered only three months, from November 1978 to February 1979. The total of cases was correspondingly reduced, being only 304 in all; but the adjusted annual rates were similar. To compensate for this relatively small sample, the effort was made to follow up each incident in depth. As it transpired, adequate follow-up was obtained for only 130 cases. The remaining 174 forms were not returned, which may or may not reflect their seriousness. Nevertheless,

a few probably valid generalisations can again be drawn. Once more, most of the inquiries originated from hospitals (90%) and most referred to children under 5 years of age (68%).

The distribution as between the various types of household cleaning products was very much as before, perhaps with the exception of "lavatory blocks," ingestion of which increased, although the overall numbers were small. It would be interesting to see if there is any relation here to sales over the period concerned. Once again the symptom picture was much the same as in 1974-5, with some two-thirds of the patients evincing no ill effects at all and the remainder suffering no more than nausea, vomiting, abdominal pain, and "burning" of the mouth. Everyone recovered satisfactorily and this time there were no deaths, though it will be remembered that the four deaths reported previously were all in adults who had deliberately swallowed excessive volumes of bleach.

Just as in 1974-5, in 1978-9 anything like major treatment was the exception and it is interesting to note the tendency for gastric aspiration and lavage to be performed less often still, no doubt because emptying the stomach in these circumstances has come to be judged of less importance and, in any event, for small children the policy has been firmly to recommend induced emesis instead.

Anyone interested in perusing the detailed tabulations is invited to write to us for a copy.

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¹ Goulding R, Jenkins HL. *Br Med J* 1978;i:286-7.

Acute renal failure after a beer-drinking binge

SIR,—We were interested to read the report of two patients developing acute renal failure following excessive ingestion of ethanol (23 February, p 526). Dr R R Ghose and others propose that muscle damage and myoglobinuria led to the development of acute renal failure. However, in neither case is clinical or laboratory evidence provided to support this claim. We have recently seen a similar patient in whom there was clear evidence of acute muscle damage associated with myoglobinuria and hyperuricaemia.

A 32-year-old unemployed barman was admitted to hospital, having been found after lying overnight at home. For the previous two weeks he had drunk one to two bottles of rum a day. On examination he was unemphatic and icteric with marked peripheral vasodilatation. His calves and buttocks were swollen, with extreme tenderness on palpation. He had evidence of mild Korsakow's psychosis and peripheral neuropathy. Blood pressure was 120/70 mm Hg. In the 12 hours following admission he passed only 50 ml of dark urine.

Blood urea was 33 n.mol/l (199 mg/100 ml) and he had a metabolic acidosis with bicarbonate 15 mmol (mEq)/l. Plasma bilirubin was elevated at 96 μ mol/l (5.6 mg/100 ml), aspartate transaminase 128 units/l. Urea-stable lactate dehydrogenase and serum creatine phosphokinase were grossly elevated at 354 and 827 units/l. Urate was grossly elevated at 1.76 mmol/l (29.6 mg/100 ml) (normal 0.12-0.42 mmol/l (2.02-7.06 mg/100 ml)). Both myoglobin and haemoglobin were detected in his urine; a haemolysis screen was negative. Following

the administration of intravenous sodium bicarbonate, his urinary output rose to 50 ml/hour. However, by the next day blood urea had risen to 38 mmol/l (229 mg/100 ml) and the urate level was unchanged. Urgent haemodialysis was performed, with biochemical improvement; in particular, postdialysis urate was 0.50 mmol/l (8.40 mg/100 ml). Following a single haemodialysis his diuresis continued over the next three days and his recovery was otherwise uneventful, with no residual renal impairment.

We agree that acute renal failure due to a nephrotoxic effect of alcohol is unlikely. In our case muscle damage was both apparent clinically and confirmed biochemically. However, the uric acid level was also grossly elevated. The mechanism of acute renal failure as a consequence of myoglobinuria is uncertain although it has been suggested that other factors, including shock and acidosis, are important.¹ It is well recognised that hyperuricaemia occurs following muscle damage.² Additionally, hyperuricaemia may lead to acute renal failure in the absence of muscle damage.³ Muscle damage may result from pressure necrosis following immobility during alcoholic stupor.⁴ We suggest that the resulting hyperuricaemia is a major aetiological factor in the development of acute renal failure in this situation.

In similar cases, priority should be given to the estimation of uric acid level and if it is grossly elevated then urgent alkalisation to increase uric acid solubility and dialysis to reduce uric acid load is indicated. In such circumstances haemodialysis is to be greatly preferred to peritoneal dialysis, as urate clearance is significantly greater.⁵

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¹ Barbanell C. In: Hamburger J, Crosnier J, Grünfeld J-P, eds. *Nephrology*. New York: John Wiley and Sons, 1979:188.

² Warren DJ, Leitch AG, Leggett RJE. *Lancet* 1975; ii:385-7.

³ Kjellstrand CM, Campbell DC, Von Hartitzsch B, Buselmeier JJ. *Arch Int Med* 1974;133:349-59.

⁴ Owen CA, Mubarak EJ, Hargens AR, Rutherford L, Gareto LP, Akson WH. *N Engl J Med* 1979;300:1169-72.

Clearance of bacteriuria on discontinuing oral contraception

SIR,—Dr D A Evans and others (19 January, p 152) conclude from their study that "clearance of bacteriuria in women who take oral contraceptives is related to discontinuation of the pill." They do not state whether the women who discontinued remained sexually active—comparability of marital status between the two groups is not a sensitive indicator of this variable. The association between sexual activity and bacteriuria is well documented; those women who stopped taking the pill may have done so because they were no longer having intercourse. Without this information the authors' conclusion is invalid.

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SIR,—I find the short report on clearance of bacteriuria on discontinuing oral contraception (19 January, p 152) interesting but would like to know: (a) what forms of contraception were substituted for oral contraceptives; and (b)

whether there was any change in the frequency of sexual intercourse on stopping oral contraceptives. Both these factors could effect the occurrence of bacteriuria.

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SIR,—Dr D A Evans and others suggest (19 January, p 152) that clearance of bacteriuria in women who have been taking oral contraceptives is related to discontinuation of the medication. One cogent reason why women stop the contraceptive pill is that they stop having sexual intercourse, an activity known to be related to the incidence of urinary tract infection and bacteriuria.¹ The authors match their patients for marital status, but in 1980 I do not think that this accurately reflects sexual activity. Perhaps the reduction in bacteriuria is at least partly accounted for by a reduction in frequency of intercourse rather than discontinuation of the pill?

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¹ Kunin CM, McCormack RC. *N Engl J Med* 1968; 278:635-42.

* * * We sent copies of the first two letters to the authors, whose reply is printed below.—
Ed, *BMJ*.

SIR,—The possibility raised by Dr Susan M Hall seems unlikely for two reasons. Contrary to her assumption that an association has been demonstrated between sexual activity and bacteriuria, we know of no adequate studies of the matter, a point that others have also emphasised.¹ Much of the suspicion that current sexual activity may be related to acquisition of urinary tract infection arises from anecdotes concerning symptomatic infections occurring soon after intercourse. Such observations are compatible with several other explanations, however, including acquisition of symptoms by women with pre-existing asymptomatic infection, the simple coincidence of infections with a frequent event, and over-reporting of striking clinical situations in a common ailment. Some support for an association may be inferred from a study² showing lower rates of bacteriuria for nuns than for women employed in the community. On the other hand, this same study and also population-based surveys³⁻⁵, including the one⁶ on which our report is based, have consistently shown that younger women in the most sexually active age groups⁶ have lower rates of bacteriuria than do older women.

Secondly, although we did not seek data on sexual activity in our study, some information may be gained from the reasons women gave for discontinuing oral contraceptives. Three of the women who stopped using them switched to another method (one an intrauterine contraceptive device, one the rhythm method, and the husband of the third had a vasectomy); one wished to become pregnant; one passed the menopause; and in two cases the reason for discontinuation was not known. Although these reasons give no quantitative estimate of sexual activity, they do not support the idea that an intent to abstain led to the discontinuation of oral contraception.

Dr Elizabeth M T Mayall raises another issue: that clearance of bacteriuria may have been related to the substitution of some new

method of contraception rather than to the actual discontinuation of oral contraception. For this mechanism to explain our findings would require that the risk of infection for women using some alternative means of contraception should be lower than that for women using no contraceptives. We know of no data supporting this idea. In addition, the variety of reasons given for stopping oral contraceptives make it unlikely that any single reason substantially modifies the association.

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¹ Kunin CM. *N Engl J Med* 1978;298:336-7.

² Kunin CM, McCormack RC. *N Engl J Med* 1968; 278:635-42.

³ Kass EH, Savage W, Santamarina BAG. In: Kass EH, ed. *Progress in pyelonephritis*. Philadelphia: FA Davis, 1965:3-10.

⁴ Freedman LR, Phair JP, Seki M, Hamilton HB, Nefzger MD. *Yale J Biol Med* 1965;37:262-82.

⁵ Evans DA, Williams DN, Laughlin WW, et al. *J Infect Dis* 1978;138:768-73.

⁶ Westoff CF. *Fam Plan Perspect* 1974;6:136-41.

Platelet-release reaction in myocardial infarction

SIR,—Dr S Heptinstall and others have demonstrated (12 January, p 80) that there is a significant difference between the platelet release reaction in patients who died within a year of myocardial infarction on the one hand and those who survived this period and control subjects on the other. This phenomenon was observed when heparin was used as anticoagulant but was masked by citrate. These observations are of considerable interest, especially if they are borne out in a larger series of patients.

We would like to suggest that the enhanced platelet release reaction in patients with poor prognosis may be related to decreased prostacyclin (PGI₂) activity. There is evidence that vessels damaged by atherosclerosis produce less PGI₂¹ and that PGI₂ may have a protective effect² (possibly including an antiarrhythmic action³) in the postinfarction period. This may account for the fact that more patients with poor prognosis had had previous infarcts than those with good prognosis.

There is evidence that heparin inhibits PGI₂ activity.⁴ Therefore a decreased PGI₂ activity could be significantly inhibited by heparin whereas higher activities in controls and patients with good prognosis would not. Furthermore, other workers have shown that in experimentally induced atherosclerosis in rabbits there is a distortion of the PGI₂: platelet thromboxane A₂ (TXA₂) ratio.⁵ More complicated mechanisms, therefore, possibly involving an increase in platelet TXA₂ activity, must also be considered.

Thus the inhibitory effect of heparin on PGI₂ may have some relevance to the observation of Dr Heptinstall and his colleagues. We would therefore like to know if the authors have investigated the effect on the release reaction of varying the concentration of heparin. Furthermore, there is also the question whether all the serotonin taken up by platelets in such an experiment is, in fact, incorporated into the same pool as endogenous serotonin.