of personality disorder and the lack of need for housewives to conform to social drinking hours may lead to a heavier—but still historically minimised—alcohol intake than in male alcoholics. The question then becomes: *for the same degree of alcohol abuse, do women fare worse than men?*

ADRIAN N HAMLYN

Department of Medicine,
Royal Victoria Infirmary,
Newcastle upon Tyne

¹ de Cruz, A G, *et al*, *Acta Hepatogastroenterologica*, 1975, 22, 369.

² Dow, J, *et al*, *Clinical Science and Molecular Medicine*, 1975, 49, 603.

³ Brenner, M H, *American Journal of Public Health*, 1975, 65, 1279.

SOMA

SIR,—Two members of the Society of Authors, both doctors, have drawn our attention to an article by Dr I J T Davies entitled "SOMA: new hope for medical authors" in your issue of 2 April (p 887).

Dr Davies says that "most medical authors get a raw deal" and that for this and other reasons a group "has now banded together to form the Society of Medical Authors." He states that "the standard contract (euphemised to 'memorandum of agreement') drawn up between most authors of medical books and their publishers was apparently agreed by the Society of Authors and the Publishers Association" (my italics). I have no idea where Dr Davies could have got this totally incorrect information. The Society of Authors has negotiated no such contract. There is, indeed, no "standard publishing contract," though some publishers tend to imply that theirs is "standard" so as to discourage authors from any attempt to bargain.

The Society of Authors has a substantial number of medical authors among its three and a half thousand members. They are entitled to individual advice on all matters connected with the marketing of their work and, in particular, to detailed "vetting" of publishing contracts. This often enables them to negotiate considerably better terms than those originally offered.

PHILIPPA MACLIESH
Secretariat,
The Society of Authors

London SW10

Paracetamol-induced acute pancreatitis

SIR,—With reference to the report by Drs I T Gilmore and E Tourvas of pancreatitis associated with paracetamol overdose (19 March, p 753), I would like to report a similar case of paracetamol overdose in which pancreatitis was associated with a lesser degree of hepato-renal failure, occurring at a different stage of the illness.

A 41-year-old machinist was admitted to hospital 48 hours after taking 25 g of paracetamol (50 Panadol tablets) with suicidal intent. She had not taken alcohol with the tablets. She had been nauseated and had vomited several times before admission. She had no abdominal pain. She had no previous history of biliary or liver disease and consumed only small amounts of alcohol.

On examination no bruising was present, she was slightly icteric but not clinically dehydrated or shocked. She had epigastric tenderness with normal bowel sounds. Initial investigations showed: Haemoglobin 15.0 g per 100 ml (15 g/dl); white cell count $6.5 \times 10^9/l$; prothrombin 20 seconds, control 13; alkaline phosphatase 125 IU/l (normal

46–190); serum glutamic oxaloacetic transaminase (SGOT) 2000 IU/l (normal 2–19); bilirubin free 45 mmol/l, conjugated 36 mmol/l, total 81 mmol/l (normal 5–17); gamma glutamyl transpeptidase 51 IU/l (normal 4–18); serum paracetamol 15 mg/ml; urea 3.4 mmol/l (normal 1.6–5).

A regimen of 50% dextrose IV giving 3000 calories a day was started controlling blood sugar with insulin via an infusion pump according to Dextrostix. Magnesium sulphate enemas and vitamin K were also given. Her initial vomiting settled with metoclopramide by the second day of admission. Her liver function tests deteriorated, with the SGPT reaching 4137 IU/l at the same time.

On the third day after admission, a further episode of vomiting occurred. She had mild abdominal pain, which worsened over the next two days, being central and radiating to the back. The abdomen became distended, with an ileus present. A diagnostic tap on the sixth day revealed turbid ascitic fluid positive for blood on ward testing with a protein content of 20 g/l and an amylase of 4000 IU. The serum amylase was 1250 IU (70–300 IU normal), calcium 1.9 mmol/l (normal 2.2–2.6), protein 55 g/l (normal 63–78), haemoglobin 12.3 g per 100 ml (12.3 g/dl), white cell count $11.5 \times 10^9/l$ with 30 000 platelets ($30 \times 10^9/l$) and urea 13.6 mmol/l. No evidence of viscus perforation on abdominal x-ray was found. A diagnosis of pancreatitis was made.

Conservative treatment of fluid replacement, analgesia, and metoclopramide resulted in some improvement. The vomiting and pain subsided by the seventh day though the serum amylase was still raised at 1000 IU/l. She remained in hospital for a fortnight, being slightly jaundiced though fairly well on discharge. The patient's liver function had returned to normal when seen in the clinic three months later and she had had no more vomiting or abdominal pain.

In this case the "pancreatitis" occurred as the liver cellular function was improving (SGPT falling) but also at a time when the alkaline phosphatase and bilirubin were climbing (especially the conjugated form) (see figure). This is in keeping with an oedematous pancreas causing an obstructive picture. The low platelet count at the time of the pancreatitis would suggest a diagnosis of disseminated intravascular coagulation, though no further tests were done to confirm this. DIC is a complication of diabetic ketoacidosis, rather than hyperglycaemia.¹ There is no evidence

that this patient was ketoacidotic, though she had episodes of hyperglycaemia. This case is presumed to be pancreatitis though retroperitoneal haemorrhage is not excluded.

Gilmore and Tourvas's case is of a lady with an alcoholic history. Though alcohol consumption with the paracetamol is not mentioned, she could have had pancreatitis associated with the alcohol.

I am grateful to Dr M S Knapp for permission to report on this patient, who was under his care.

R A COWARD

Salford Royal Hospital,
Salford

¹ Timperley, W R, *et al*, *Lancet*, 1974, 1, 952.

Therapeutic test with colchicine in diagnosis of gout

SIR,—It was interesting to read the experience of Dr M L Snaith and Dr E N Coomes in gout with normal serum urate concentrations (12 March, p 685). They emphasise that a definite diagnosis of gout cannot be made unless monosodium urate crystals are seen in the joint aspirate. They conclude by referring to the value of a therapeutic test with colchicine as a diagnostic aid in acute gout (presumably when polarised light microscopy facilities are not available). I wish to point out that the arthritis of sarcoidosis,^{1,2} psoriasis,³ erythema nodosum, and pyrophosphate arthropathy⁴ also responds to colchicine. Although bearing in mind that sarcoidosis and psoriasis can be accompanied by hyperuricaemia, it appears to me that a therapeutic test with colchicine in the diagnosis of acute gout is unreliable and probably should be abandoned.

P KINSELLA

Department of Rheumatology,
Middlesex Hospital,
London W1

¹ Kaplan, H, *New England Journal of Medicine*, 1960, 263, 778.

² Kaplan, H, *New England Journal of Medicine*, 1963, 268, 761.

³ Bunim, J J, *Annals of Internal Medicine*, 1962, 57, 1018.

⁴ Huskisson, E C, *Lancet*, 1972, 2, 271.

Interviewing women applicants

SIR,—At several recent job interviews I have been struck by the proportion of the (brief) time available which is spent discussing the present employment and career prospects of my husband (also a doctor) rather than my own. My husband, on the other hand, has never had his marital status so much as mentioned in a similar situation. I am sure all married women are well aware of the problems of combining marriage and career, but it is sometimes difficult to avoid the conclusion that one is regarded as an appendage to the husband who has no need of a job anyway. I should be interested to know whether any of your readers have similar experiences or feelings on this subject.

CHRISTINE M GASTON

Wembley Hospital,
Wembley, Middx

Increased university fees and graduate medical students

SIR,—May we add our voice to that of the graduates at Southampton Medical School