that, in some instances at least, the truth may be as closely approached by those housemen who record only a vague descriptive comment (or even no comment at all).

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¹ Orrego H, Blendis LM, Blake JE, Kapur BM, Israel Y. *Lancet* 1979;ii:1354-6.

SIR,—The study in your last issue by Dr I G Barrison and others (18 October, p 1040) incorporated two false premises: (a) that "heavy drinkers" are likely to reveal themselves during "routine history taking"; and (b) that a heavy drinking habit is a necessary or sufficient condition for the diagnosis of an alcoholic.

Further, one-third of the patients studied were emergency admissions. It is, therefore, hardly surprising that immediately non-relevant details, such as drinking habits, might have been overlooked in the wish to make a diagnosis and institute appropriate treatment in the very sick patient. In routine admissions—for example, for dental treatment—a drinking history might well seem out of context both to patient and houseman. It is a credit to the housemen at Charing Cross that only a relevant 15% of the patients had not been asked about drinking habits (that is the ratio of those non-teetotallers who had not been asked to the population studied).

Alcoholics are the very people that are most likely to give false drinking histories. Furthermore, in the absence of explicit admission of heavy drinking or requests of help from patient or family, it is unclear what effective action might be carried out for those heavy drinkers discovered during "routine history taking" in hospital.

There is a negative attitude to alcoholism, as the authors point out. This raises the question of who is in the best position to diagnose likely alcoholics. This must be a person who has intimate knowledge of the patient, his family, and his environment. Thus, only the general practitioner and other members within the community can be expected to substantially increase the diagnosis of the problem. It is their education on alcoholism, not the houseman's, that is of primary importance.

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Malignancies and false-positive serological results for hydatidosis

SIR,—Some authors,^{1,2} including Dr F van Knapen recently in the *BMJ* (19 July, p 195), have reported false-positive serological results for hydatidosis in patients with malignancies.

In our laboratory serological diagnosis for hydatid disease is routinely carried out by simplified counterimmunoelectrophoresis on cellulose acetate membrane with a lyophilised antigen, commercially produced for the complement fixation test (Behring, L'Aquila, Italy, code OTMN 05), as described in detail elsewhere.³ This method, in our experience, is positive in hydatidosis only.

Briefly, antigen is dissolved in 0.5 ml distilled water and is frozen (at -20°C) and thawed (at 26°C) five times. The cellulose acetate membrane (Chemetron, Milan, Italy, code 077) is immersed for 10 minutes in tris-glycine buffer, pH 9.5 (Chemetron, code 067), dried with blotting

paper, and mounted on to the plastic bridge. Each sheet is sufficient for six tests. Five microlitres of antigen are placed on the cathodic side and 5 μl of serum on the anodic side of the membrane. Counterimmunoelectrophoresis is carried out in tris-glycine buffer at 200 V for 35 minutes in an ELVI 20 apparatus (ELVI, Milan). The cellulose acetate strip is then washed four times for 15 minutes in saline with continual stirring, stained in coumassie blue (Chemetron, code 080) for 15 minutes and destained (decoloring solution Chemetron, code 092) until the precipitation bands are clearly observed. In positive sera the arcs of precipitation are generally in the form of an "upper lip" or a thick and undulating streak.

With this method we have examined sera from patients with surgically confirmed hydatidosis (69 sera), other parasitic diseases (35), malignancies of the lung (22) and liver (17), blood disorders including myeloma, leukaemia, lymphoma, etc (15), and normal individuals (50). Positive results occurred with sera from patients with confirmed hydatidosis (in 56 cases—that is, 81%) but not with any other sera.

Our method, if confirmed by other workers, could be therefore useful in the differential diagnosis of space-occupying masses. Clearly the most likely diagnosis should always be made on the basis of all available clinical and laboratory data.

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¹ Yong WK, Heath DD, Savage T. *Br Med J* 1979; i:1463-4.

² Varela-Diaz VM, Coltorti EA, D'Alessandro A. *Am J Trop Med Hyg* 1978;27:554-7.

³ Mansueto S, Migneco G, Tripi S, Piccone DM. *Trans R Soc Trop Med Hyg* 1980;74:260-1.

When is it justified to stop the pill?

SIR,—I supervise contraception for a number of patients in accordance with DHSS guidelines. I have been concerned that four of my patients have recently been taken off the pill by others without my consent. I accept that this is necessary for major surgery to the pelvis, but these were young women undergoing surgery to bunions or extraction of wisdom teeth by local dentists. One patient became pregnant and had an abortion.

Comment from the local academic department of gynaecology has been noncommittal. However, to stop low-dose oestrogen pills for these sorts of very minor operations on young women seems to me to be wrong. Perhaps through your columns I may ask if surgical, dental, or family planning colleagues have seen enough cases of thrombosis and embolism in such circumstances to warrant the risk of unwanted pregnancies. In 10 years of general practice I have seen none.

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Cimetidine and ranitidine in duodenal ulcer

SIR,—Professor M J S Langman and others (16 August, p 473) conclude that "cimetidine and ranitidine give equivalent results in patients with duodenal ulcer." At week 4, 72% of cimetidine-treated patients had healed and

63% of ranitidine treated patients had healed; this is a difference of 9% in favour of cimetidine, but with only 18 patients in one group and 19 in the other the 95% confidence interval for the difference in the percentage of patients healed while on cimetidine versus those healed while on ranitidine runs from 40% to -25% .¹ In other words, subsequent studies could show a difference between cimetidine and ranitidine as great as that between cimetidine and placebo. A study with 20 patients per group will fail about half the time to show a significant difference between two treatments for which the true difference is 30%.

This study suggests that cimetidine and ranitidine may prove to give equivalent results in future tests but does not allow the conclusion that they will.

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¹ Peterson WL, Elashoff JD. *Gastroenterology* 1980;79: 585-8.

Myopathy associated with cimetidine?

SIR,—The description by Dr T J Walls and others (11 October, p 974) of two cases of motor neuropathy possibly associated with cimetidine has prompted us to report the following case.

A 71-year-old man began treatment with cimetidine (1 g daily) in the summer of 1977 for a barium-meal-proved duodenal ulcer. In August 1978 he rapidly developed painless weakness of hip and shoulder girdles such that he was unable to get out of bed or comb his hair. Other than long-standing (20 years) Raynaud's phenomenon affecting his hands, there were no other symptoms, and he was on no other medication. He had smoked 15 cigarettes daily for many years. On admission to hospital three months later general examination was normal; power was markedly reduced in all four limbs proximally (MRC grade 3-4) though not distally, with a similar distribution of wasting; sensation was normal; the lower limb tendon reflexes were absent and those in the upper limbs reduced. The following tests were normal or negative: chest radiograph, barium meal, routine haematology and biochemistry, creatinine phosphokinase, mid-stream urine examination, triiodothyronine, tetraiodothyronine, Wasserman and Venereal Disease Research Laboratory tests, and serum auto-antibody screens. Protein electrophoresis showed a slight increase in α_1 - and α_2 -globulins, a random blood sugar was 13.6 mmol/l (245 mg/100 ml) with a fasting level of 7.6 mmol/l (137 mg/100 ml), and the erythrocyte sedimentation rate (ESR) 59 mm in the first hour. Motor nerve conduction velocities in upper and lower limbs were within normal limits and coaxial needle electromyograms of forearm, hand, lower leg, and foot muscles normal. Electromyograms from deltoid and quadriceps showed a low voltage myopathic pattern with no fibrillation potentials or positive sharp waves.

The cimetidine was stopped and over the subsequent year he made a remarkable recovery. His mild maturity-onset diabetes has remained under excellent control on glibenclamide 2.5 mg daily and the ESR has fallen to 10 mm in the first hour. No evidence for any neoplastic lesion or arteritic process has appeared in the two years of follow-up. Because of a return of his ulcer symptoms he has recently recommenced cimetidine in a dosage of 200 mg twice daily without any demonstrable effect on muscle power so far.

Although muscle pains are recorded with cimetidine therapy¹ to our knowledge there are no recorded cases of an associated myopathy. Until Walls *et al* had demonstrated a possible neurotoxic property of cimetidine we were reluctant to believe that this was the causal factor in our patient. The timing of the