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Comatose patients smelling of alcohol

SIR,—We welcome your leading article highlighting the regular nightmare confronting doctors working in accident and emergency departments.[1] It is hardly surprising that a patient with a head injury is the commonest serious surgical admission on any day of the week in this region.

We have dealt with large numbers of patients with head injuries, most of whom have been drinking. More recently, ingestion or injection of drugs has created additional diagnostic problems in patients presenting in coma and smelling of drink. We urge caution in looking after patients who have no skull fracture on radiography and who have a low blood alcohol concentration yet remain in coma. They may have taken almost any substance, including opiates, hypnotics, sedatives, analgesics—that is, whatever is available in the "market." Many of these patients are brought to accident and emergency departments in police custody, and the police are understandably reluctant to take them back into custody without a clear cut diagnosis having been made.

Most patients seen in accident and emergency departments are looked after by junior hospital doctors. What should they do? On the one hand they are harangued by radiologists who complain that too many skull radiographs are ordered and on the other hand by neurosurgeons who rightly urge prompt referral to a neurosurgical centre when appropriate. We suspect that our new managers, armed with the results of clinical audit, will join the chorus and complain of too many "unnecessary" admissions.

We need more diagnostic help. We need a readily available, accurate imaging technique to detect intracranial problems reliably while the patient is in the accident and emergency department. We need a readily available and safe antagonist to alcohol to transform the problem of alcohol overdosage like naloxone has transformed opiate overdosage. Most of all we need a more responsible attitude to alcohol by all sections of society. In the end, several problem drinkers will still present a disproportionate workload to accident and emergency departments, and we fear that nothing that has been done or said in the past will have any effect on this group.

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1 Quaghebeur G, Richards P. Comatose patients smelling of alcohol. *Br Med J* 1989;**299**:410. (12 August.)

SIR,—I am most impressed that Dr Gerardine Quaghebeur and Mr Peter Richards have an emergency blood alcohol service available to them[1]; I would guess that most casualty departments are not so fortunate. In these hospitals useful information can be gained by calculating the "osmolar gap"—a means of estimating the concentration of low molecular weight substances (such as alcohol) that are present in large amounts. This approach is not mentioned by Dr Quaghebeur and Mr Richards, and I suspect that their silence reflects a gap in knowledge that is common to many clinicians.

In brief, the approach is to measure the plasma concentrations of sodium, postassium, urea, and glucose; an estimate of plasma osmolarity can be derived from these. A variety of formulas have been suggested, and a useful example is[2]: calculated osmolarity=(sodium concentration+potassium concentration)×1·86+urea concentration+glucose concentration.

The result is then compared with the actual osmolality (as measured by the laboratory). Small differences (of about 10 mmol/l) can be accepted as due to the approach's limitations. A blood alcohol concentration of 40 mmol/l, however, would increase the measured osmolality by about 40 mmol/l compared with the calculated value. Only a few other substances have the same effect, the most common of these being methanol and ethylene glycol.

Some hospitals do not measure ethanol concentrations "in house"; those that do often use gas chromatography, which is time consuming and demands skilled staff. Estimating the osmolar gap is quick and requires only those facilities that are already available in all district hospitals. It deserves to be more widely understood by casualty officers.

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1 Quaghebeur G, Richards P. Comatose patients smelling of alcohol. *Br Med J* 1989;**299**:410. (12 August.)
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SIR,—In their editorial Dr Gerardine Quaghebeur and Mr Peter Richards deal with some very relevant points regarding treatable conditions in comatose patients smelling of alcohol[1]; but I was disappointed not to see any mention of a further treatable condition in such patients—namely, Wernicke's encephalopathy. Although coma in this condition is thought to be rare[2,3] and its onset insidious,[4] it is extremely important that it is considered and treated early as death and serious neurological sequelae occur commonly.[3,5]

I have experience of three patients admitted to medical and surgical wards with histories of chronic alcohol abuse who became severely neurologically impaired as a result of Wernicke's encephalopathy not being diagnosed or treated. The signs in a comatose patient may be confused with, or put down to, the effects of a brain stem cerebrovascular accident or head injury. The signs thought to be helpful in diagnosing the condition are preserved pupillary light reflexes, absent focal signs, and absent caloric and doll's eye responses.[2] Hypothermia[6] and hypotension[5] may be present, and a lack of tendon reflexes due to peripheral neuropathy[4] is also helpful. The recommended treatment is 50 mg of thiamine given parenterally followed by 50 mg three times daily for several days initially.[4]

Although relatively uncommon as the sole cause of coma or, more commonly, decreased conciousness in comatose patients smelling of alcohol, the serious and severe consequences of not treating Wernicke's encephalopathy quickly make it mandatory to give any patient considered to be a risk sufficient thiamine acutely, and usually parenterally. This may prevent the unnecessary and disabling consequences of the disease

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Diagnosing rubella

SIR,—The editorial by Dr Peter Morgan-Capner on diagnosing rubella[1] has prompted us to write of our recent experience with a case of rubella reinfection in pregnancy.

A 25 year old woman in her second pregnancy gave a history of contact with a patient with clinically diagnosed rubella and had herself developed a rash in the fifth week of her pregnancy. Rubella specific IgM, detectable by a capture radioimmunoassay, was found in two serum samples taken four and six weeks after the clinical illness. We substantiated this as a reinfection by analysing a serum sample collected eight months before during her previous pregnancy along with the current serum samples. Rubella antibodies, at a concentration consistent with immunity, were detected in all three serum samples by three tests (latex agglutination, single radial haemolysis, and haemagglutination inhibition), and a rise in total antibody concentration was confirmed between the sample taken before the illness and those taken afterwards. Thus with a serologically confirmed clinically apparent reinfection we were able to advise the patient and offer other diagnostic services based on chorionic villus sampling. The outcome of the pregnancy is still to be determined.

We agree with Dr Morgan-Capner that a single positive result of "rubella antibodies present" should not be treated as infallible, particularly if a single method has been used. Our case illustrates the importance of archival material, which allows confirmation of previous results and the performance of further tests in difficult cases. Serum samples should be stored for as long as practicably possible. The case also shows that the presence of rubella antibody does not necessarily indicate immunity to clinical disease. Indeed, a Public Health Laboratory Service working party has recently recommended that laboratories should desist from making this interpretation.[2] All rashes in pregnancy should be investigated for evidence of rubella infection regardless of previous tests.

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Use of endoscopy in patients with dyspepsia

SIR,—Dr Dino Vaira and colleagues found that 58% of patients with dyspepsia without ulceration had evidence of gastritis and *Campylobacter pylori* on histological examination.[1] They have, however, not provided the age distribution of their patients. In the general population the prevalence of the organism rises with increasing age, reaching about 50% in those over 50.[2] Hence the prevalence of *C pylori* in their patients is possibly no different from that expected.

Their conclusion that endoscopy is a poor indicator of histological gastritis and the presence of *C pylori* in patients without ulcers has been well documented.[3-7] It therefore seems unnecessary to compare it with other tests that specifically aim at diagnosing the presence of *C pylori*. The authors

also seem to presume a cause and effect relation between the presence of *C pylori* gastritis and dyspepsia. The evidence that gastritis causes dyspeptic symptoms is controversial.[1] Gastric biopsy is essential for diagnosing *C pylori* gastritis, but whether it should be performed routinely in all patients with dyspepsia without ulceration is another matter. Should gastritis and the presence of *C pylori* in such patients be regarded as causing their symptoms and an indication for treatment with regimens to combat *C pylori*? Until the answer is available any suggestion for performing routine gastric biopsy in patients with dyspepsia without ulceration should be regarded as premature.

Their conclusion that "endoscopy is unhelpful in patients with dyspepsia if endoscopic biopsy specimens are not taken routinely" is also misleading. As they state, most organic lesions that account for dyspepsia are found at endoscopy. Although biopsy is essential for diagnosing *C pylori* gastritis, we believe that routine endoscopic biopsies in patients with dyspepsia without ulceration should be recommended only if patients are being entered into controlled trials.

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AUTHORS' REPLY,—In reply to Dr R Upadhyay and colleagues, our patients had a median age of 43 years.

We agree that the association of *Campylobacter pylori* with gastritis and the sensitivity of endoscopy for detecting gastritis have been documented; as Dr Upadhyay and colleagues state, however, the relation between *C pylori* associated gastritis and dyspepsia without ulceration is not clear and has not been well documented. The stated aim of our study was to understand this relation and not to compare endoscopy with specific tests for *C pylori*. We gave the sensitivity and the specificity of each test as they are relevant information.

We find it difficult to understand how Dr Upadhyay and colleagues could believe that we presumed a cause and effect relation when we clearly stated: "It is not clear how commonly gastritis associated with *C pylori* occurs in dyspepsia without ulceration and whether the presence of *C pylori* results in different symptoms." The first question is clearly: Is *C pylori* associated gastritis a cause of dyspepsia without ulceration? The answer will become available only by looking for *C pylori* in patients with this condition and appropriate controls. The second question is: How reliable is endoscopic appearance in detecting gastritis? We calculated how often endoscopy could detect gastritis and believe that "eye balling" the antral and gastric mucosa is unacceptable as it has a positive predictive value of only 52% and a negative predictive value of 38%. Gastritis is not of course necessarily the cause of the symptoms, but it is only by performing such studies that this will be determined. We therefore undertook a double blind placebo controlled prospective trial, which is nearly completed, in patients who have dyspepsia without ulceration and who are normal on endoscopy but have abnormal histological features. On the other hand, in a non-trial setting, we believe that it is reasonable to give such patients a therapeutic trial of appropriate treatment, perhaps even including psychiatric assessment, rather than further invasive investigation.

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SIR,—In their description of 107 dyspeptic patients in whom endoscopy showed no organic lesion Dr D Vaira and colleagues made the not entirely surprising discovery that the sensitivity, specificity, and predictive values of histopathological tests are superior to those of endoscopic appearances in diagnosing conditions that are invisible to the naked eye.[1] These authors go on to make the extraordinary and unsupported statement that endoscopy is unhelpful in patients with dyspepsia if endoscopic biopsy specimens are not taken routinely.

What do these authors mean by helpful? Do they mean that a research study of the prevalence of gastritis associated with campylobacter cannot be made without histopathological examination? This may not be true; the urease breath test is probably accurate enough for these purposes. Do they mean that the clinician is unable to formulate an appropriate management plan without full histopathological information? There is very little evidence to show that the clinical course or symptomatic response in non-ulcer dyspepsia is affected by distinguishing between a normal and an inflamed mucosa. Although Rokkas *et al* have reported a small, short term study of the beneficial effects of bismuth treatment on symptoms in dyspepsia negative on endoscopy,[2] longer term studies suggest that the clinical course of this condition is benign and that most patients recover spontaneously.[3]

In a further study of 226 dyspeptic patients with negative results of endoscopies who were followed up for a minimum of two years we were able to confirm that non-ulcer dyspepsia follows a benign clinical course and that in most cases the symptoms resolve either spontaneously or after short term treatment with antacids or antisecretory drugs. Important gastrointestinal events during the follow up period were rare and affected only five patients. Consultation rates for gastrointestinal problems in the study group fell significantly after endoscopy (from 2·6 (SD 1·7) per year to 0·97 (1·3) per year, $p<0·001$), and there was also a substantial fall in drug prescribing, both for gastrointestinal drugs (2·1 (1·9) to 0·97 (1·5) prescriptions per patient per year, $p<0·001$) and for all drugs (4·7 (4·2) to 3·6 (3·6), $p<0·01$).

Do Dr Vaira and colleagues mean that the endoscopy is unhelpful for the patient? We are given virtually no information about the patients or the clinical context in which this study was conducted and have no idea whether the histological findings influenced subsequent management and outcome. It seems likely, for instance, that a substantial number of the patients in whom abdominal bloating was a prominent symptom had some kind of functional bowel or motility disorder rather than having symptoms directly related to their gastritis. Without follow up information about the impact of the histopathological findings on recovery and relapse of symptoms, statements about the helpfulness of the investigation to the patient are meaningless.

The implied recommendation in this paper is that endoscopic biopsy in all cases of non-ulcer dyspepsia is mandatory. Perhaps Dr Vaira and colleagues should pause to consider that, on the basis of figures obtained in one hospital with a sophisticated resource management system in the endoscopy unit (C W Venables, personal communication), adding biopsy procedures to a standard oesophagogastroduodenoscopy increases the cost of the procedure by about £73. If treatment with acid suppressing agents or bismuth is then recommended as a result of these findings, for which the clinical correlates remain spurious, the hidden cost of an already unnecessary investigation escalates further.

Performed and reported in isolation from a recognisable clinical context and with no reference to relevant published reports, this study provides only a partial view of the subject; a comparison of clinical outcomes in normal endoscopies with and without routine biopsy might clarify the issue.

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Risks of AIDS among workers in the "sex industry"

SIR,—It may well be that "homosexual prostitutes are, in many large cities, not far inferior in number to the females who are engaged in heterosexual prostitution,"[1] but in our recent review of published work we found only 10 scientific papers on male prostitution that had been published since 1980. The report by Ms R M Thomas and colleagues[2] is therefore welcome as it considerably increases knowledge on this subject, although despite the title what they estimated was the risk of HIV infection, not AIDS.

Nobody knows how many sex workers there are in any society, and attempts at categorising behaviour patterns have often been bedevilled with cultural bias, population selectivity, and inability to corroborate independently reported behaviour patterns. The "sex industry" includes not only people who provide sexual services for money but also the unknown, probably large, number of men and women who offer sexual services in return for drugs, food, accommodation, etc. This group is particularly difficult to study and probably does not use existing services well. The data of Ms Thomas and colleagues, for example, show that only 109 of 205 respondents had sought medical advice over an unspecified period, highlighting the need to provide services targeted at this group. At our clinic we run specific clinics for both male and female sex workers, offering various services in conjunction with outreach organisations. In our experience male sex workers seem to be less likely to attend clinics than their female counterparts.

We believe that "snowballing" as a method of