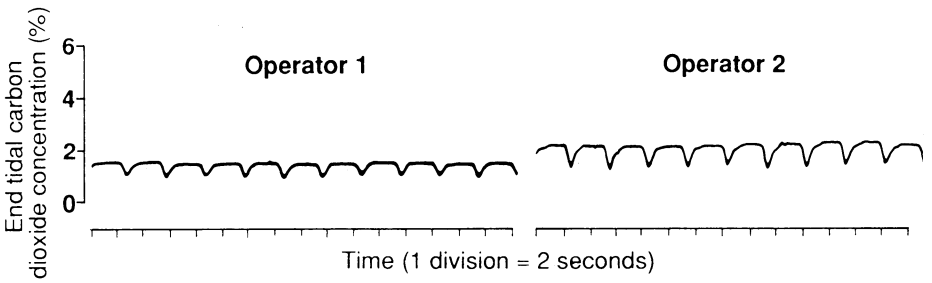

FIG 1—*Infrared analyser trace showing end tidal carbon dioxide concentration with two different physicians giving external chest compression*

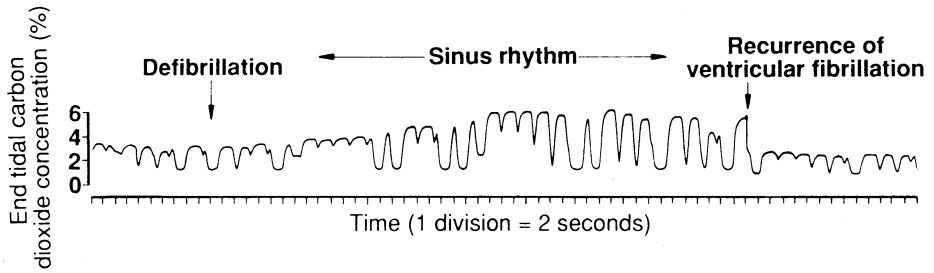

FIG 2—*Infrared analyser trace showing changes in end tidal carbon dioxide concentration with successful defibrillation and with recurrence of ventricular fibrillation*

circumstances. We have studied 13 adults who had suffered cardiac arrest and required endotracheal intubation and cardiopulmonary resuscitation. The lungs were ventilated with 100% oxygen by using a Motivus 2 portable ventilator delivering a constant minute volume, and end tidal carbon dioxide concentration was measured with a Godart Model BE infrared analyser.

During external chest compression the mean (SD) end tidal carbon dioxide concentration was 2·23 (0·92)%. When chest compression was stopped to check for a spontaneous circulation it fell to 1·57 (0·86)%. During each phase the end tidal concentration was calculated for each patient by taking a mean value from five consecutive breaths. This difference was significant (paired Student's t test, $p<0·01$). We also noted the rapid and sustained rise in end tidal concentration associated with the return of a spontaneous circulation, when it reached a mean of 5·08 (1·48)%.

The technique provides an instantaneous and continuous guide to the efficacy of external chest compression and, as Dr Higgins and colleagues noted, will indicate when this is inefficient. Figure 1 shows the rise in end tidal concentration that occurred (from 1·53% to 2·39%) in one patient when chest compression was taken over by a more experienced and vigorous physician.

Equally helpful was the monitoring of the abrupt fall in carbon dioxide concentration that was seen with any serious failure of the circulation once it had been restored. Figure 2 shows the increase in end tidal concentration after successfully defibrillating a patient in ventricular fibrillation and then the rapid fall as ventricular fibrillation recurred.

Although giving sodium bicarbonate produced no consistent discernible change in end tidal concentration, in some cases it was injected into a peripheral vein and the lack of effect may have reflected the considerable delay in its reaching the central circulation and the tissues thereafter. Others have found the rise in end tidal concentration after giving bicarbonate to be short lived and far less than that seen when spontaneous circulation is restored.[2]

Previous workers have studied patients either arriving in the accident and emergency department[2] or already in intensive care units.[3] Our study and that of Dr Higgins and colleagues show that this technique can be applied equally well in general wards. The studies confirm that monitoring end tidal carbon dioxide concentration provides a practical and non-invasive means of assessing the circulation during cardiopulmonary resuscitation. As such it forms a valuable guide for the resuscitation team and a useful teaching aid for those learning these techniques.

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1 Higgins D, Hayes M, Denman W, Wilkinson DJ. Effectiveness of using end tidal carbon dioxide concentration to monitor cardiopulmonary resuscitation. *Br Med J* 1990;**300**:581. (3 March.)
2 Garnett AR, Ornato JP, Gonzalez ER, Johnson EB. End-tidal carbon dioxide monitoring during cardiopulmonary resuscitation. *JAMA* 1987;**257**:512-5.
3 Falk JL, Rackow EC, Weil MH. End-tidal carbon dioxide concentration during cardiopulmonary resuscitation. *N Engl J Med* 1988;**318**:607-11.

Dangers of thrombolysis

SIR,—With reference to the leading article by Mr M C Petch concerning the early use of thrombolytic agents in acute myocardial infarction[1] I would like to report the following case history.

I am a general practitioner in an isolated rural practice, which is 40 km from the nearest district general hospital and 11 km from the nearest ambulance station. I was called to see a patient—a 51 year old businessman (who was normotensive, overweight, and a former smoker)—at 3 am. He had suffered three days of epigastric discomfort, which had suddenly worsened and become a crushing low chest pain with breathlessness, which was not helped by sublingual glyceryl trinitrate. He did not describe any other symptoms.

His medical history included the occurrence of an anteroseptal infarct in June 1986. Investigations at that time showed a fasting cholesterol concentration of 8·9 mmol/l and triglyceride concentration of 2·2 mmol/l, and he weighed 96 kg (body mass index 31·4 kg/m^2). He was discharged on a low cholesterol and weight reducing diet and taking metroprolol 50 mg daily. He stopped smoking but continued to suffer occasional episodes of chest pain, although it was not clear whether these were due to angina. On review in April 1987 his fasting cholesterol concentration was 7·5 mmol/l, so he was given bezafibrate 400 mg daily, which reduced his cholesterol concentration to 4·86 mmol/l. His alcohol intake was in excess of 20 units per week. He had no history of peptic ulceration.

On examination he was apyrexial, dyspnoeic, distressed, pale, and sweaty. His pulse was regular at 84 beats/min and blood pressure was stable at 120/70 mm Hg. There were no signs of cardiac failure, and I found no evidence of acute abdominal disease. Being unsure of the diagnosis I withheld anistreplase and performed electrocardiography, which showed no acute changes. I gave him morphine 15 mg and oxygen. Thankfully, despite the dispute, the local ambulance crews were providing an emergency service, and a call direct to Maldon ambulance station achieved an immediate response.

After eight minutes my patient's condition deteriorated rapidly; he had severe crushing retrosternal chest pain and epigastric pain. I therefore gave him diamorphine 5 mg and anistreplase 30 units intravenously as I had little doubt that he was having a myocardial infarction.

I travelled with him to hospital (a journey taking 40 minutes) but thankfully he remained stable and his pain gradually subsided. Further electrocardiography performed after admission to the coronary care unit showed no acute changes, and his enzyme activities were later found to have been normal. As he had clearly not had an infarction and as he subsequently developed a fever he was treated with intravenous cefuroxime for presumed cholecystitis and his care was transferred to the members of the surgical team who were on call; they could find no evidence of a surgically amenable cause. An ultrasound scan and oral cholecystogram failed to show any gall bladder abnormality, and he has remained well since discharge.

I think that the fever was probably an adverse reaction to the anistreplase, and both the patient and I are convinced that the anistreplase prevented an impending infarction. If fibrinolysis had been withheld until admission the outcome might have been quite different. I advocate that all rural general practitioners should keep thrombolytic agents and be prepared to use them, but the present arrangements for paying general practitioners for the cost of these expensive drugs make this unlikely.

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1 Petch MC. Dangers of thrombolysis. *Br Med J* 1990;**300**:483-4. (24 February.)

Genital warts

SIR,—I was interested in the lesson of the week by Dr C A Carne and Mr G Dockerty,[1] having recently published a similar study.[2] Our study was carried out prospectively at the department of genitourinary medicine at Guy's Hospital during the latter part of 1988. Unlike Dr Carne and Mr Dockerty we considered only female patients. We too felt that it was appropriate to reassess the observations of Kinghorn[3] in the light of the availability of testing for *Chlamydia trachomatis* infection.

Whereas Dr Carne and Mr Dockerty found that 18·5% of women had coincident infection with *C trachomatis* we found by a similar technique that only 9% were positive. One of these women positive for chlamydia was also infected with *Trichomonas vaginalis*; there were no other cases of sexually transmitted infection among our patients.

A similar rate of *C trachomatis* infection (10·7%) has been found in a general practice population.[4] Fish *et al* found that 9·5% of women aged 16-25 attending a gynaecology clinic were infected with chlamydia.[5] We therefore concluded that among our female patients with genital warts the incidence of other sexually transmitted diseases was no higher than among women of similar age within the community. Clearly this was not so among the Cambridge patients. There must have been some intrinsic difference between the two groups of

patients. In inner London most women with warts refer themselves to the genitourinary clinic. Attendance is easy as little travelling is necessary; a high rate of attendance is therefore likely. It might be that in the Cambridge clinic with a larger catchment area women would be less likely to attend a department of genitourinary medicine unless other factors (such as recent change of partner) made them more concerned that they might harbour other diseases. Local policies for referral, screening, and treatment of patients with genital warts need to be tailored to local conditions; such policies need to be based on audit such as that carried out by Dr Carne and Mr Dockerty and ourselves.

In their introduction Dr Carne and Mr Dockerty mention "the link between the aetiological agent of warts, human papillomavirus, and the development of cervical cancer." The causative agent in genital warts is human papillomavirus type 6 or 11.[6] Types 16 and 18 have been implicated in the aetiology of cervical cancer[7] but types 6 and 11 have not. There is therefore no reason to anticipate an association between genital warts and cervical cancer. Despite this it seems to be a widespread policy to advise women with genital warts that they have an increased risk of this malignancy and to carry out more frequent cytological screening in them.

No study has shown any increased risk of cervical cancer in women with vulval warts. On the contrary, there have been three reports of case-control studies[8-10] that have shown that warts do not represent a risk factor for cervical cancer.

It is time to stop telling women with warts that they have an increased risk of cervical cancer and to stop the wasteful overscreening of these women.

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1 Carne CA, Dockerty G. Genital warts: need to screen for coinfection. *Br Med J* 1990;**300**:459. (17 February.)
2 Griffiths M, Overington LJ, Chard SC. Genital warts and the need for screening. *Genitourin Med* 1989;**65**:399.
3 Kinghorn GR. Genital warts: incidence of associated genital infections. *Br J Dermatol* 1978;**99**:405-9.
4 Longhurst LJ, Flower N, Thomas BJ, *et al*. A simple method for detection of Chlamydia trachomatis infection in general practice. *J R Coll Gen Pract* 1987;**37**:1255-6.
5 Fish ANJ, Fairweather DVI, Oriel JD, Ridgway GL. Chlamydia trachomatis infection in a gynaecology clinic population. *Eur J Obstet Gynecol Reprod Biol* 1989;**31**:67-74.
6 Gissmann L, Villiers E-M, zur Hausen H. Analysis of human genital warts and other genital tumours for HPV type 6 DNA. *Int J Cancer* 1982;**29**:143-6.
7 Muñoz N, Bosch X, Kaldor JM. Does HPV cause cervical cancer? The state of the epidemiological evidence. *Br J Cancer* 1988;**57**:1-5.
8 La Vecchia C. The epidemiology of cervical neoplasia. *Biomed Pharmacother* 1985;**39**:426-33.
9 La Vecchia C, Franceschi S, Decarli A, *et al*. Sexual factors, venereal diseases and the risk of intraepithelial and invasive cervical neoplasia. *Cancer* 1986;**58**:935-41.
10 Slattery ML, Overall JC, Abbott TM, *et al*. Sexual activity, contraception, genital infection and cervical cancer: support for an STD hypothesis. *Am J Epidemiol* 1989;**130**:248-58.

SIR,—We agree with the principle of screening patients with genital warts attending genitourinary clinics for other sexually transmissible infections[1] but would also screen those with anal and perianal warts, conditions that may constitute the only clinical manifestation of human papillomavirus infection.[2] Further, it was not clear whether the patients screened for sexually transmitted disease presented with symptoms associated with their warts (a relatively rare event[3]) or whether they presented for other reasons and were found on clinical examination to have warts, in which case the proportion of those with conditions in addition to their warts would be increased. Even less clear is the position regarding contact tracing. We have no doubt that attempts should be made to trace the contacts of patients who have, in addition to their warts, a treatable sexually transmitted disease. The rationale of tracing the contacts of those who have only clinically apparent human papillomavirus infection is, however, dubious. The purpose of contact tracing is to identify those who have acquired the infection from the index case and to treat them. Making the association may be difficult because the use of modern technologies has highlighted the widespread carriage of human papillomavirus in clinically normal subjects,[4] in women with cervical cytological abnormalities,[5] in unexpected sites,[6] and in those who would not normally be regarded as at particular risk of having sexually transmitted diseases, such as neonates.[7]

The problem is not just one of dealing with overt warts but of determining the existence of non-apparent human papillomavirus infection and then of what to offer the contact. There is currently no established treatment for asymptomatic human papillomavirus infection and no evidence that conventional methods of treatment can reliably eradicate it even in patients with visible warts. The frequent recurrence of clinical disease regardless of the mode of treatment[3] illustrates the difficulty of managing this condition. It is not inconceivable that, like some other viral conditions, human papillomavirus infections may be lifelong and that treatment may be purely cosmetic. Whether or not to advise the use of condoms and if so, for how long, is unanswered. Only when the natural course of human papillomavirus infection and its interrelation with clinically apparent and non-apparent lesions have been clearly defined by adequate longitudinal and epidemiological studies can we develop rational treatment and offer soundly based advice on this poorly understood condition.

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1 Carne CA, Dockerty G. Genital warts: need to screen for coinfection. *Br Med J* 1990;**300**:459. (17 February.)
2 Oriel JD. Anal warts and anal coitus. *Br J Vener Dis* 1971;**47**:373-6.
3 Tyring SK, Cauda R, Baron S, Whitely RJ. Condyloma acuminatum: epidemiological, clinical and therapeutic aspects. *Eur J Epidemiol* 1987;**3**:209-15.
4 Young LS, Bevan IS, Johnson MA, *et al*. The polymerase chain reaction: a new epidemiological tool for investigating cervical human papillomavirus infection. *Br Med J* 1989;**298**:14-8.
5 Campion MJ, McCane DJ, Cuzick J, Singer A. Progressive potential of mild cervical atypia: prospective cytological and virological study. *Lancet* 1986;ii:237-40.
6 McDonnell JM, Mayr AJ, Martin WJ. DNA of human papillomavirus type 16 in dysplastic and malignant lesions of the conjunctiva and cornea. *N Engl J Med* 1989;**320**:1442-5.
7 Roman A, Fife K. Human papillomavirus DNA associated with foreskins of normal newborns. *J Infect Dis* 1986;**153**:855-61.

Implications of inflammatory changes on cervical cytology

SIR,—We were surprised to note that Dr J D Wilson and colleagues detected *Chlamydia trachomatis* in the cervical specimens of 18% of their patients with inflammatory changes on cervical cytology.[1] (The women had been referred by general practitioners and community family planning clinics.)

We studied retrospectively a similar series of women with inflammatory changes on cervical cytology. They had been referred by family planning clinics in Wandsworth Health Authority to the department of genitourinary medicine at St George's Hospital between 1 May 1987 and 30 April 1988. We detected *C trachomatis*, which was identified by using direct immunofluorescent monoclonal antibody (Microtrak, Syva, California, United States), in the cervical specimens of only three out of 98 women, which is a similar figure to that found by others.[2] The detection rate of *C trachomatis* in the cervical specimens of women attending the department because they were a sexual contact of a man with non-specific urethritis during the same period was 25%. Only one patient had *Trichomonas vaginalis* infection and one had genital warts, which is in agreement with the findings of Dr Wilson and colleagues.

We think that a larger study would be necessary to ascertain the prevalence of chlamydial infection in women with cervical smears that show inflammatory changes before recommendations as to the management of such women can be made.

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1 Wilson JD, Robinson AJ, Kinghorn SA, Hicks DA. Implications of inflammatory changes on cervical cytology. *Br Med J* 1990;**300**:638-40. (10 March.)
2 Toon PG, Arrand JR, Wilson LP, Sharp DS. Human papillomavirus infection of the uterine cervix of women without cytological signs of neoplasia. *Br Med J* 1986;**293**:1261-4.

Alcoholic doctors can recover

SIR,—Dr Gareth Lloyd and his colleagues in the North West Doctors and Dentists Group are to be congratulated on their courage and wisdom in attempting to evaluate the outcome of their experiences as doctors with a severe alcohol problem and in publishing their findings.[1]

In the past we have been faced with uncertainty as to how these groups work and as to their effectiveness. This difficulty has arisen because of the importance the profession attaches to confidentiality. Until this curtain of secrecy is lifted and the profession learns that there is hope for these doctors and their families then treatment, support, and rehabilitation will always be too little and too late.

Dr Lloyd's study has confirmed that alcoholic doctors can recover. This, too, has been the General Medical Council's experience: over one third of severely disabled doctors referred to its health machinery return to professional practice. To achieve an even better result the disease has to be identified earlier and treated before it has destroyed the doctor and his or her family and reputation. This requires a determination by the profession to provide a more effective local machinery than is currently available. The responsibility lies with the profession, not the General Medical Council. The motivation would be humanity, necessity, and conviction.

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1 Lloyd G. Alcoholic doctors can recover. *Br Med J* 1990;**300**:728-30. (17 March.)

Intratesticular radionuclide and spermatogenic damage

SIR,—The recent statistical analysis of data on the incidence and distribution of cases of acute lymphoblastic leukaemia around Sellafield[1] has stimulated several controversial hypotheses concerning causative factors.[2] Paternal exposure in high risk areas of the reprocessing plant is one suggestion, either by external irradiation or from internally incorporated isotopes, which could cause internal continuous low dose irradiation of the gonads with a potential for damage for which there are currently no relevant published experimental data.

During the past three years we investigated cell targeted indium-114m to the lymphoid system as a radiotherapeutic regimen for treating leukaemia.[3,4] This entailed extensive tissue distribution studies and whole body clearance of the radioisotope over prolonged periods in a rat model of T cell