mide-alcohol flushing (and indomethacin) as predictors of future development of vascular disease.

The loss of the differential association between chlorpropamide-alcohol flushing and macroangiopathy in diabetics diagnosed more than 15 years ago underlines the need for cautious interpretation. In table II of the paper by Drs Barnett and Pyke the patients without chlorpropamide-alcohol flushing of both age ranges are unusual in their greater liability to macroangiopathy with the shorter duration of diabetes; as so often, one wonders how patients entered a particular study and what a "total population" study would show, though realising how arduous this would be. The fixity of the chlorpropamide-alcohol flushing phenotype is an underlying assumption of the interpretation of Drs Barnett and Pyke, but its all-or-nothing character has already been questioned (by Jefferys *et al* at a recent meeting of the British Diabetic Association's medical and scientific section), while Köbberling has failed to confirm via cheek temperature measurement any increase of chlorpropamide-alcohol flushing-positivity among maturity-type onset diabetics compared to non-diabetics or even to juvenile-type onset patients.

Finally, what are the substantial reasons to postulate any "role of prostaglandins in the aetiology of both (vascular) types of complications"[1] from the observations that (1) maturity-onset diabetics with chlorpropamide-alcohol flushing and without angiopathy have the flush blocked by indomethacin; (2) indomethacin acts to inhibit a prostaglandin synthetase; (3) intravenous infusion of prostaglandin E is followed by facial flushing; and (4) absence of chlorpropamide-alcohol flushing is associated with increased risk of angiopathy? It is reminiscent of last year's involvement of endorphins[3] in the aetiology of maturity-type onset diabetes.

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[1] Barnett AH, Spiliopoulos AJ, Pyke DA. *Lancet* 1980; ii:164-6.
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[3] Leslie RDG, Pyke DA, Stubbs WA. *Lancet* 1979;i: 341-3.

## Bladder cancer as a prescribed industrial disease

SIR,—The correction (16 August, p 523) to Dr F J Darby's letter (17 May, p 1229) on this topic, altering his statement that "Because the carcinogens responsible for causing urothelial tumour were withdrawn from industrial use in the early 1950s, the number of cases should show a decline over the next decade" (p 1230) to "by the mid-1960s . . ." makes the statement neither true nor any less misleading to clinicians and others who have to help certain sufferers from urothelial tumours (or their widows or widowers) to obtain such benefits as they may be entitled to under Prescribed Industrial Disease Regulations No 39.[1] In any case the correction is incomplete, for "over the next decade" must also be altered by adding some 15 years to this time because of the long latent period (averaging about 20 years) which is involved.

Perhaps it may help to restate the situation. Dr Darby's letter, which because of the address on it will be deemed to express the official view of the DHSS, was a comment on a paper by Sheena Somerville and others entitled "Bladder cancer as a prescribed industrial disease: a guide for clinicians" (23 February, p 540), which gave help in defining under what circumstances a claim under PD 39 regulations might succeed. If it is to do so two criteria must be fulfilled—firstly, the disease must be of the right sort and, secondly, the patient must at some time after 4 July 1948 have been in insurable employment in a situation where the right chemicals were manufactured, used, or liberated, even if the employment consisted merely in maintaining or cleaning the plant used or laundering clothes used by workers in some such plants if this was done professionally in or for the plant concerned. It is not necessary, in relation to any particular case, that exposure reached or exceeded any particular level (see the appeal of G E Rushworth in 1979,[2] where a tribunal commissioner stated in judgment, "I agree with what was stated by a commissioner in decision R I 2/77 'that the presence of even very small quantities of a relevant substance is sufficient for the prescription of PD 39.' "). Nor is it necessary in any particular case to show that the disease was in fact causally related to the occupation in relation to which PD 39 benefit is claimed.

The "relevant substances" are set out in the regulations[1] in a form not easily understandable by those who are not chemists; but Dr Darby's phrase "the carcinogens responsible for causing urothelial tumour" will, in my opinion, inevitably be construed as being synonymous and coextensive with the PD 39 list; and because of the "authority" given to it by the letters DHSS clinicians will be tempted to ignore a patient's working history after "the mid-1960s" when considering whether to advise that PD 39 benefit should be sought.

In fact, far from a decline in the number of instances where such benefit should be sought—as opposed to any diminution of the total number of cases of occupational bladder cancer achieved by the growing control of the conditions of manufacture and use of the at present recognised carcinogenic aromatic amines since about 1950—an increase in patients entitled to benefit is to be expected. This is because it is not, on the whole, patients who have been employed in the larger manufacturing and using industries who stand in need of advice, for their employers and unions tend to take care of this, but because of new situations where exposure to the "relevant substances" occurred are coming to light as scientific knowledge increases. Unfortunately such knowledge takes a long time to reach clinicians (and apparently the DHSS) and so needy patients or their families do not get such recompense as they are in fact entitled to.

One such example is the discovery a few years ago that the induline and some nigrosine dyestuffs as manufactured in this country contained about 0·8% of 4-aminodiphenyl,[3] a carcinogen at least as potent as 2-(β-)naphthylamine in humans. Although this level of impurity just keeps it out of the "prohibited substances" class of the Carcinogenic Substances Regulations 1967[4] it should be remembered that the 2-(β-)naphthylamine impurity in the rubber antioxidant Nonox S, blamed as the main cause of the severe outbreak of occupational bladder cancer in the British rubber industry,[5] was about 0·2%. Induline has uses in the dyeing industry; in the rubber and plastic industry; in making varnishes and stains; and in making typewriter ribbons, carbon paper, and inks—to quote but a few examples. How many sufferers have as yet been advised to make claims relating to the use of induline dyes or products made from them and containing some of the 4-aminodiphenyl?

If, as Dr Darby contends, the urothelial carcinogens had indeed been withdrawn by the mid-1960s, why was it necessary to pass the Carcinogenic Substances Regulations,[4] which did not cover all the "relevant substances" listed in the PD 39 regulations (but which dealt with no other substances), and which prohibited only a few of them, in December 1967? Why, in January 1979, did the Health and Safety Commission[6] find it necessary to send a warning letter to several British chemical suppliers pointing out the possible illegality of their offering for sale "certain substances specified as Prohibited Substances in Part II of the Regulations."[1]?

Could, and would, Dr Darby or some other official of the DHSS issue an authoritative list of where the "relevant substances" occur in industry or products, and of the dates, if any, when they were withdrawn? If not, clinicians and other advisers, as suggested in Sheena Somerville's paper, will still have to seek advice from sources other than the DHSS.

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[3] Parkes HG. *British Rubber Manufacturers' Association Health Research Unit Bulletin* 1977; No 20.
[4] Carcinogenic Substances Regulations 1967: Statutory Instruments 1967, No 879. London: HMSO, 1967.
[5] O'Connor Mr Justice. *Cassidy T and Wright C v Dunlop Rubber Co Ltd and ICI Ltd: Judgement.* London: High Court of Justice, Queen's Bench Division, 1971:25.
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## Sauna-induced acceleration in insulin absorption

SIR,—Dr H J Cüppers and others (26 July, p 307) have responded to my article (14 June, p 1411), in which I reported a significant rise in $^{125}$I-insulin absorption (as measured externally by gamma counter) in diabetic patients after a Finnish sauna. In their study the authors injected healthy subjects subcutaneously with 10 U of Actrapid insulin prior to sauna (30 min at 85°C) and determined the levels of serum immunoreactive insulin following the injection. As compared with the control day, no increase in serum insulin levels was observed after the sauna. On the basis of their failure to see a rise in serum insulin levels after sauna and on previous findings in animals suggesting local degradation of insulin at the injection site,[1] Dr Cüppers and his colleagues criticise the method of external measurement of insulin disappearance from the injection site employed in my study.

If, however, one compares my results with their data more carefully, there is no discrepancy in the results: I also failed to see any significant rise in insulin absorption after the first 25 minutes of sauna. It was only after a repeat 25 minutes of sauna that insulin absorption in diabetic patients was significantly higher than on the control day at room temperature. More recently we have demonstrated that when ambient temperature was raised from 20° to 35°C diabetic patients demonstrated a 50-60% increase in insulin absorption over a four-hour period.[2] This rise in insulin absorption was accompanied by a 25% increment in skin temperature ($p < 0.05$) and a 2·3-fold increase in skin blood flow ($p < 0.05$). These observations, in conjunction with our results during the sauna, clearly demonstrate an enhanced insulin absorption in diabetic patients exposed to heat for 50-240 minutes.

Regarding the claim that the external measurement of insulin disappearance from the injection site ". . . is not suitable to assess the circulating levels of exogenous insulin following its subcutaneous injection," a variety of studies have demonstrated that (*a*) the radioactivity registered externally correlates with the amount of extractable insulin at the injection site[3]; (*b*) in human studies a correlation has been observed between the disappearance rate of $^{125}$I-insulin from the injection site (as measured by external detection) and the change in blood glucose determined at rest[3–5] or during exercise.[6] Thus the available data indicate that the external measurement of the fall in radioactivity at the site of insulin injection reflects the disappearance rate of $^{125}$I-labelled rapidly

acting insulin and suggests that the absorbed insulin is biologically active.

On the other hand, measurement of plasma insulin levels in healthy subjects may not accurately reflect insulin absorption kinetics under all circumstances. Firstly, circulating levels of plasma insulin in healthy people are the sum of endogenous plus exogenous insulin and do not reflect the amount of absorbed insulin alone. This is important, since exogenous insulin is shown to decrease endogenous insulin secretion in the normal person.[7] Secondly, serum insulin levels are affected not only by the rate of insulin delivery but also by the rate of insulin clearance. Factors which may affect insulin clearance regardless of their effect on insulin absorption may affect plasma immunoreactive concentrations after insulin injection. Furthermore, owing to the rapid turnover of insulin, plasma insulin levels at a given time represent only a minor portion of subcutaneously injected insulin.[8 9] Thus under circumstances of altered endogenous insulin secretion or changed metabolic clearance of insulin the determination of serum insulin levels as an index of insulin absorption kinetics is subject to error.

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[2] Koivisto VA, Fortney S. *Diabetes* 1980;suppl 2:26A.
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[4] Koivisto VA, Felig P. *Ann Int Med* 1980;**92**:59-61.
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## Jejunoileal tuberculosis

SIR,—In their report on jejunoileal tuberculosis, Dr Caroline Humphreys and others (12 July, p 118) state that the differential diagnosis of Crohn's disease and gastrointestinal tuberculosis is difficult and requires a high index of suspicion. It is my opinion that the diagnosis could have been entertained if the purified protein derivative (PPD) reactivity had been tested prior to starting therapy with corticosteroids. The time-honoured practice of testing patients before immunosuppression has to be emphasised because in similar situations it has a two-fold purpose: firstly, it brings up the possibility of gastrointestinal tuberculosis[1] and, secondly, will determine need for isoniazid prophylaxis.[2]

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[1] Sherman S, Rohwedder JJ, Ravikrishnan KP, Weg JG. *Arch Intern Med* 1980;**140**:506-8.
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## Acupuncture and postherpetic neuralgia

SIR,—We would like to add to the reply to your question about acupuncture (26 July, p 283). One of us (GL) has treated about 20 patients suffering from established postherpetic neuralgia with acupuncture. Many of these had had invasive or destructive procedures prior to the acupuncture treatment, but despite this approximately 40% seem to have gained significant pain relief from acupuncture.

We realise, however, that this is no more than a clinical impression. We are establishing a trial to prove, or disprove, the efficacy of acupuncture in postherpetic neuralgia. This trial will be starting towards the end of this year and will draw patients from the Southampton area. Acupuncture will be compared with an equally magical placebo, and the effects will be assessed by a non-treating doctor (JF). We would be happy to provide protocols for doctors who are interested, and would be grateful for criticism of our methodology.

We feel that the case for steroids and other antiviral agents in the treatment of either shingles or the prevention of postherpetic neuralgia is unproved. In China acupuncture is given to all patients suffering from acute shingles, and this may explain the extremely low incidence of postherpetic neuralgia. Perhaps it is more reasonable to suggest that patients should receive a course of acupuncture during the acute shingles?

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## Medical audit

SIR,—I agree with Sir Cyril Clarke (16 August, p 514) that the Senior Hospital Medical Staff Conference's rejection of the CCHMS's proposals for medical audit should not be interpreted as hostility to the principles of quality control. There are certainly many hospitals in the UK in which regular case discussions, clinicopathological conferences, and death reviews are a firmly established feature of the postgraduate scene. I would depart from Sir Cyril's remarks only in emphasising that such reviews have in many instances been going on for more than a quarter of a century and not simply, as he suggests, since the initiation of the Medical Services Study Group. But I have little doubt that that group has given encouragement to many physicians.

I am sure that collaborative research can be a valuable promoter of healthy self-criticism and that it should be more widely encouraged on a regional and national basis. The Royal College of General Practitioners has done some useful work on these lines, and so has the Medical Services Study Group of the Royal College of Physicians of London, for which Sir Cyril and his colleagues have been responsible. The Royal College of Surgeons of England has received encouraging financial support for developments on similar lines: many of us are hopeful that this will lead to collaboration with the specialist associations and other interested groups and individuals.

The essence of success in collaborative research, and indeed in all forms of quality control, is that it should stem from the free and willing co-operation of enthusiastic doctors. One can persuade, but should never compel, the unwilling and the sceptical to join in. If this concept of collaborative research between consenting adults in private should seem too weak a potion for those who talk so glibly of compulsory audit, let it be clearly recognised that any politically inspired and bureaucratic "big brother" would be wholly inimical to promoting the economics of excellence in medicine.

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## Advertisement from group of homosexual doctors

SIR,—The *BMJ* recently refused to publish an advertisement from a group of homosexual doctors. The aim of the advertisement was to contact other homosexual doctors to discuss matters of mutual concern. The decision was defended at the Newcastle Annual Representative Meeting, on the grounds that it would be offensive to most doctors reading the journal. The fact that no one spoke in public in favour of the advertisers might be taken to indicate the absence of any doubt about the issues involved. It is conceivable that potential speakers were worried about the personal consequences of being branded as homosexual themselves. It is clear that the discussion was rather limited.

It needs to be pointed out that a similar group existed in 1976-7, based at the University of London Union. After a year's painstaking work this group produced a document describing counselling facilities for homosexual people in distress. It then distributed the document to over 10 000 general practitioners in London. Is this kind of work really offensive?

That there are homosexual doctors must be accepted universally. If it is this that gives offence the choices are clear: the profession must go on being offended indefinitely, or attempt to remove all homosexual doctors from practice. I do not believe that this is the aim of the BMA. It seems on the surface that offence is really given by the thought of homosexual doctors talking to each other and publicising their existence. What prompts this reaction, which is at the same time harsh and dishonest? It must be that the offence that has been given to homosexual doctors (and patients) is either unrecognised or discounted. Is it surprising in this atmosphere that some homosexual doctors pretend to be heterosexual and that some homosexual patients are wary of doctors in general?

I do not believe that the majority of doctors are offended by the thought of such an advertisement. In fact, I believe that a great many might find the issue rather uninteresting. So it would be if only the journal had published the advertisement, and if only the profession had not been misrepresented in public by appearing to express unanimous disapproval. Ultimately, though, if doctors could stop being offended by each other in this way we could all get on with the work of medicine.

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## Dumfries and Galloway: where the NHS works well

SIR,—I have read with much pleasure the article by a "Special Correspondent" (9 August, p 438). I write in reply because I had the good fortune to be in Dumfries for one year. In October 1930 I became assistant to the "legendary practitioner Dr Gordon Hunter." I can thus vouch that he "provided