As retinal changes usually precede the development of diabetic nephropathy, in the five patients with proteinuria but without retinopathy (three positive and two negative for chlorpropamide-alcohol flushing) the proteinuria may possibly have been due to other causes. If these cases are excluded the difference between the groups positive and negative for flushing becomes greater (1/191 (0·5%) *v* 12/100 (12%).

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Requests for reprints should be sent to Dr D A Pyke, Diabetic Department, King's College Hospital, London SE5 9RS.

[1] FitzGerald MG, Gaddie R, Malins JM, O'Sullivan DJ. Alcohol sensitivity in diabetics receiving chlorpropamide. *Diabetes* 1962;**11**:40-3.
[2] Leslie RDG, Pyke DA. Chlorpropamide-alcohol flushing: a dominantly inherited trait associated with diabetes. *Br Med J* 1978;ii:1519-21.
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Chlorpropamide-alcohol flushing and microangiopathy in insulin-dependent diabetes

Patients with non-insulin-dependent diabetes often show facial flushing after drinking alcohol when taking chlorpropamide (chlorpropamide-alcohol flushing).[1 2] We found that patients who show this reaction have a decreased frequency of retinopathy[3] and proteinuria[4] compared with those who do not.

In view of the association between chlorpropamide-alcohol flushing and diminished risk of microangiopathy in non-insulin-dependent diabetics we wondered whether the same was true for insulin-dependent diabetics. We therefore examined the frequency of chlorpropamide-alcohol flushing in an unselected group of insulin-dependent diabetics (group 1) and two selected groups of patients with (group 2) and without (group 3) microangiopathic complications.

Patients, methods, and results

The criterion for insulin dependence was that the patient had been taking insulin from the first few weeks after diagnosis or had shown appreciable ketonuria. In group 1 the general frequency of chlorpropamide-alcohol flushing in insulin-dependent diabetics was studied by testing patients chosen at random from our diabetic clinic. Group 2 comprised patients who had developed severe microangiopathic complications within 10 years of the diagnosis of diabetes—namely, maculopathy with visual deterioration, proliferative retinopathy, or any form of retinopathy with persistent proteinuria. Group 3 comprised patients who, after more than 15 years of diabetes, showed no diabetic complications. The mean ages of the patients (±SD) at diagnosis of diabetes in groups 2 and 3 were 23 ± 15 and 22 ± 14 years respectively. The mean durations of diabetes were 22 ± 8·5 and 26·6 ± 8·6 years respectively.

Each patient was given a placebo to take on the first day of the test and then 40 ml of sherry 12 hours later. On the second day chlorpropamide 250 mg was given and the sherry again taken 12 hours later. The patient was not aware that a placebo was being used. Patients were asked to report any unusual sensation in the face within 45 minutes of consuming the alcohol. Results were analysed by the χ^2 test.

Of the 125 unselected patients in group 1, 20 (16%) showed chlorpropamide-alcohol flushing. A further five patients flushed with both placebo and chlorpropamide and were excluded. Of the 72 patients with severe complications (group 2), six (8%) showed chlorpropamide-alcohol flushing. None showed flushing with the placebo tablet. Of the 117 patients with no complications (group 3), 33 (28%) showed chlorpropamide-alcohol flushing. A further three also flushed with placebo and were excluded. The difference in the frequency of chlorpropamide-alcohol flushing between groups 2 and 3 was significant ($p < 0.01$), as was the difference between groups 3 and 1 ($p < 0.02$). The difference in frequency between group 1 and 2 did not reach significance.

Comment

In view of the relatively low frequency of chlorpropamide-alcohol flushing in insulin-dependent diabetics—16% in this series—the design of this study was different from that in our previous studies in non-insulin-dependent diabetics.[3 4] We thought that the best way to establish differences in the frequency of chlorpropamide-alcohol flushing between insulin-dependent diabetics with and without complications was to look at groups selected from each extreme—namely, those in whom severe microangiopathy had developed in the early years after diagnosis and those with no complications after many years of diabetes. There was a distinct difference in the incidence of flushing between the two groups (9% *v* 28%); the frequency in the unselected group lay between these two extremes (16%). Thus, as in non-insulin-dependent diabetics,[3] chlorpropamide-alcohol flushing in insulin-dependent diabetics seems to be associated with a decreased frequency of microangiopathy, though the association is not so striking. We have not yet done any family studies of chlorpropamide-alcohol flushing in insulin-dependent diabetics so we do not know whether it is an inherited feature in such patients, as we think it is in non-insulin-dependent diabetics,[2 3] and thus whether there is an inherited component to the development of diabetic retinopathy in insulin-dependent diabetes.

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Group R streptococcal meningitis (Streptococcus suis type II): a new industrial disease?

Four cases of meningitis due to group R streptococci occurring in men working with pigs or pork products have been described in England. I report a fifth case and suggest that this infection should be classed as an industrial disease.

Case report

A 61-year-old meat inspector was referred to this hospital in coma. Six days earlier he had inspected a consignment of pig carcasses, which were subsequently condemned. He had not accidently cut himself during this inspection, and no routine bacteriological cultures had been taken from the carcasses. Four days later he developed a sore throat with fever and shivering, which resolved within 36 hours. The day before referral he had noticed pain at the base of his back, followed 12 hours later by a gradual onset of headache, vomiting, and difficulty in hearing. From this time his level of consciousness steadily deteriorated.

On admission he was comatose, restless, with pronounced nuchal rigidity, and a fever of 40°C. There was no sign of cutaneous infection, and examination of fauces and external ears showed no abnormality. Appearances on funduscopy were normal, and there were no abnormal focal neurological signs. Lumbar puncture showed turbid cerebrospinal fluid under raised pressure; microscopical examination showed 18×10^3 white blood cells/μl (95% polymorphs) with 36 red blood cells/μl, and abundant Gram-positive cocci. Cerebrospinal fluid protein concentration was 4 g/l and glucose was undetectable. Treatment was immediately started with ampicillin, 200 mg/kg/day, given as 4 g six-hourly intravenously. Twenty-four hours later benzylpenicillin, three megaunits six-hourly intravenously, was added when