

diet histories. This group of men can all be regarded as essentially lactose aborbers, since less than 3% of the population in southern Sweden is lactose intolerant.¹ The daily lactose intake ranged between less than 1 g and nearly 100 g.

We selected for eye examination 25 men with the highest lactose intake (mean 17.2 g per 1000 kcal, range 13.8–33.3), one middle group of 25 men (8.5 g per 1000 kcal, range 3.5–13.6), and the 25 men with the lowest lactose intake (mean 1.70 g per 1000 kcal, range 1–3.5). Eyes were examined, with pharmacological dilatation of the pupil to reveal any small traces of cataract, by a doctor who did not know to which lactose group the man belonged.

Minor cataract changes were observed in 29% of the high lactose group, in 44% of the middle group, and in 40% of the low lactose group. These figures do not indicate a positive correlation between high lactose intake and early cataract—rather the reverse. We do not yet know for certain whether these early changes are related to later fully developing cataracts. Of these 75 men only 1 had been operated on for cataract, and he was in the low lactose group (1.5 g per 1000 kcal). We plan to continue this investigation by looking at lactose intakes in all men with fully developed cataracts in the whole group (n = 621).

Our conclusion for the present is that we have no evidence for the hypothesis that the amount of lactose consumed in lactase persistent individuals affects the prevalence of early cataract changes, but we are not yet ready to state whether there is any relation between lactose intake and clinically fully developed cataract.

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OPERATIVE STRATEGY FOR PHAECHROMOCYTOMAS LOCALISED BY [¹³¹I]-m-IODOBENZYLGUANIDINE SCINTIGRAPHY

SIR,—The adrenomedullary scanning agent [¹³¹I]-m-iodobenzylguanidine (MIBG) is very useful in the localisation of phaeochromocytomas. Dr Brown and colleagues (Jan 7, p 56) reported a patient with a right adrenal phaeochromocytoma in whom the phaeochromocytoma and the normal left adrenal medulla were demonstrated five days after injection of the isotope. Normal adrenal glands are rarely seen at 24 h (and only faintly when they are) but the frequency of visualisation increases over the ensuing days.² Furthermore, nearly all phaeochromocytomas can be visualised on scanning after 24 h. Therefore, the misinterpretation of the five-day scan by Brown and colleagues should not be regarded as a false-positive result, and we concur with the comments of Dr Chatal and colleagues (March 31, p 733). In our experience with more than 450 patients who were suspected of having a phaeochromocytoma there were no false-positive scans, with a scanning dose of 0.5 mCi MIBG (one-quarter of the dose used by Brown et al).

Of more concern to us is the unilateral approach to an apparently localised phaeochromocytoma advocated by Brown and colleagues. No localisation technique is absolutely reliable. The two best investigations, which will suffice in nearly all circumstances, are computerised tomography and MIBG scanning, and these have complementary roles.³

We have treated a patient in whom MIBG scanning revealed a definite phaeochromocytoma in the left adrenal only; at laparotomy through a curved transverse incision bilateral adrenal medullary

hyperplasia (of the sporadic type) with bilateral adrenal phaeochromocytomas was found, and there was a third phaeochromocytoma arising in paraganglionic tissue anterior to the abdominal aorta. This patient would not have been cured by a unilateral left flank approach.

While computerised tomography and MIBG scanning are of major assistance in the planning and performance of operations for phaeochromocytomas, they provide no justification for abandoning the standard surgical approach which should include transabdominal exploration of both adrenal glands and the retroperitoneal locations of extra-adrenal phaeochromocytomas (paragangliomas).

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PROLIFERATIVE RETINOPATHY AND DIABETES MELLITUS

SIR,—We have read with interest the report by Dr Knight and colleagues (March 24, p 681) of an optic nerve infarction and improvement in diabetic control after the continuous subcutaneous infusion of insulin (CSII) in a patient with insulin-dependent diabetes.¹ But, as Dr Kohner points out (May 5, p 1018), this patient's optic nerve infarction occurred when his diabetic control (though improved) was still far from adequate.

We have recently seen a patient in whom rapidly progressive proliferative retinopathy developed within 8 weeks of the institution of tight diabetic control. This 31-year-old woman with insulin-dependent diabetes was admitted to hospital with diabetic ketoacidosis and possible subarachnoid haemorrhage (excluded later by normal CT scan and CSF), having neglected her diabetes for the previous 9 years. She had had intermittent treatment with oral hypoglycaemic agents and insulin during this period, but was essentially symptom-free until an influenza-like illness precipitated ketoacidosis. After treatment of the ketoacidosis, she was started on twice daily injections of a combination of insulins. Blood glucose was maintained at 4–7 mmol/l and her HbA_{1c} had fallen from 14% to 7.5% after 8 weeks. At this stage, she complained of blurred vision and was found to have advanced proliferative retinopathy, exudates, haemorrhage, and macular oedema bilaterally. After fluorescein angiography, laser treatment was started and the macular oedema subsided and her vision stabilised.

The rapid development of proliferative retinopathy within 8 weeks of the institution of tight diabetic control and the absence of the condition despite 9 years of badly controlled diabetes suggests a causal relation between tight diabetic control and proliferative retinopathy. In view of the previously reported, though less striking, deterioration of retinopathy after tight diabetic control with CSII,¹ the possibility that rapid, sustained falls in blood glucose may aggravate retinopathy has to be considered.

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OPTIC DISC SWELLING IN DIABETES MELLITUS

SIR,—Dr Lavin and Dr Goran's restricted definition of papilloedema (March 17, p 629), is not universally accepted by clinicians. Only on etymological grounds would it seem reasonable to use the word to describe oedematous swelling of the optic disc arising from a variety of causes.² We do, however, agree that such swelling results from an interruption of axoplasmic flow in acute ischaemic optic neuropathy (AION) and other conditions.

Whether ischaemic damage in AION involves the optic nerve proximal or distal to the lamina cribrosa is uncertain. Although longitudinal capillary anastomoses are present in the optic nerve, there is no evidence that the pial vessels can maintain an adequate

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