Dr Anne Charlton reported on a survey of 15 000 schoolchildren aged 9-19 in Cumbria and Tyne and Wear.[3-7] She found that most experimentation with cigarettes starts at between 9 and 11, regular smoking usually starts at about 12 or 13, and children are more likely to smoke if their parents do so. Other factors that increase the chances that children will smoke are parental approval; brothers and sisters, friends, and teachers who smoke; advertising; and beliefs about the perceived "benefits" of smoking. Similar results were reported in a study from Derbyshire by Holland and his group, who concluded that any health education programme must start before 14 to stand a chance of success.[8]

Guidelines for health education on smoking have been developed based on data from these and similar studies, and Dr Charlton is currently assessing the impact of a new education programme on smoking.[9] The Brigantia smoking prevention programme employs a step by step process that starts at 9 and continues until 17 and encourages the children to share and discuss the information with their parents. Preliminary data suggest that this approach can decrease smoking among boys and their fathers but has less effect in girls and their fathers. Mothers, disturbingly, showed no swing away from smoking.

Any effects of passive smoking are particularly important as even a small increase in risk of lung cancer or other diseases will affect most of the population. Wald *et al* have concluded from a recent meta-analysis that people exposed to passive smoking have a small but definite increased risk of developing lung cancer.[10] In addition, the children of smokers have a lower weight at birth, show poorer growth, and are more prone to respiratory problems.[11,12] Though changes in smoking policies in British workplaces have been slow, the Health Education Authority has just produced a booklet, *Smoking Policies at Work*, and there appears to be public support for change. In a MORI poll of 854 workers in 1986 only 14% believed that smoking should be allowed in all workplace areas, while 80% would have agreed to some degree of restriction. Any changes should be planned and include participation and agreement from staff at all stages of their development. Such a policy has proved successful in several British companies, and pressure for change in others is likely to grow. Further government legislation on smoking in public places is contemplated. In 1985 the London Underground introduced a smoking ban, a change which produced little resistance from the travelling public. Clearly some forms of legislation are likely to be acceptable to the public.

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Management of retinal vein occlusion

Occlusion in the retinal venous system is a common cause of visual loss in the middle aged in the United Kingdom.[1] Occlusion of both the central and the branch veins reduces the visual acuity, but the severity of this varies. Even when the central retinal vein is occluded, however, the visual loss occurs slowly, over hours or even days, so that the patient may only inadvertently become aware of the deficit, for example, when occluding the unaffected eye. New blood vessels may develop in the iris in 20-50% of patients with central retinal vein occlusion with the invariable development of neovascular glaucoma. Until the mid-1970s retinal vein occlusion and its complications (which also include macular oedema) were regarded as untreatable. Nevertheless, the encouraging recent results obtained from treatment with laser photocoagulation has prompted an aggressive approach towards neovascularisation. Given, moreover, that the mortality rate in patients with retinal vein occlusion from cardiovascular disease (particularly stroke) is double the national average and that 10-15% of patients develop a further retinal venous occlusive event, often resulting in blindness, there is also now an emphasis on a combined prophylactic approach by both a physician and ophthalmologist.[2,3]

We still do not know what causes retinal vein occlusion, though recent data suggest that there are several factors. Histological studies support the concept that it is due to damage to the endothelium, with proliferation of the endothelial cells the major primary histological change; this may be associated with degeneration, phlebosclerosis, or secondary intramural thrombus formation.[4,5] Conditions that predispose to retinal vein occlusion include vasculitis, chronic glaucoma, diabetes mellitus, hyperviscosity syndromes, and use of contraceptives containing oestrogen.[6-8] Nevertheless, these factors are present in only a few patients, and hypertension (often poorly controlled) is the most frequently associated condition, being present in half the middle aged patients.[9] Hyperlipidaemia is found in just under a third of middle aged patients and is the predominant condition found in young patients, indicating that it may play an important part.[3,9] Other abnormal test results include raised concentrations of C reactive protein and immunoglobulin A, as well as an increased plasma viscosity and erythrocyte sedimentation rate.[10,11] Abnormal platelet aggregability and in vivo platelet function have been reported even in patients with no other underlying disease, suggesting that platelet aggregation might be an important sequel to endothelial swelling, thus leading to the occlusion.[12,13] Statistically, also, more of these patients smoke and drink than people without retinal vein occlusion.[7,9,14]

Clinical management, then, consists of treatment of the ophthalmic complications, together with systemic investigation and medical treatment of any abnormalities that are found. Retinal photocoagulation may inhibit the formation of new blood vessels or bring about regression, thereby preventing vitreous haemorrhage. In the central form treatment of the ischaemic retina may additionally inhibit the development of rubeosis of the iris, so preventing neovascular glaucoma.[15-17] Although macular oedema after occlusion of the branch veins may benefit from focal laser treatment, after occlusion of the central vein the results are disappointing.[18]

A wide variety of initial medical treatment has been tried to improve retinal venous flow but the results have been unrewarding.[6 19-23] Long term management should centre on the implications of underlying cardiovascular disease—in particular, the identification and treatment of hypertension is paramount, given the evidence that treatment may reduce the severity of some of its complications.[24] A similar approach to the treatment of hyperlipidaemia is also justified.[25]

Patients with hypertension, hyperlipidaemia, and low HDL-cholesterol concentrations are probably most at risk of recurrence and need intensive medical treatment to try to prevent this. In our view use of controlled studies of the effects of such treatment is difficult to justify on ethical grounds in view of the important prognostic implications of hypertension and hyperlipidaemia with respect to coronary and cerebrovascular disease. And our preliminary results with an aggressive therapeutic regimen aimed at individual abnormalities have been encouraging: in 400 patients presenting consecutively over five years with a single episode of retinal vein occlusion and treated intensively there was only a 1% rate of recurrence compared with the 10-15% in historical series.

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Testing paternity: traditional methods usually adequate

Recent publicity on using DNA fingerprinting—a genetic identification technique—to settle paternity problems has led to many inquiries from patients in paternity disputes. We are concerned that many doctors do not seem to understand the potential of the long established blood grouping tests to settle disputes: in 97-99% of cases they can provide proof of non-paternity where the father has been wrongly named. Additional tests are then unnecessary. In one in six cases submitted to two centres in 1986 proof was provided that the putative father was not the child's biological father.

Blood group testing also offers valuable evidence that helps to establish paternity in many cases. When it cannot be proved that the putative father is not the father then a mathematical assessment is made of the chance that he is the father. One international system is the paternity index, which is the ratio of the chance the putative father has of producing in one sperm the genes required to father the child to the chance of him doing so if he is unrelated to the child. The index gives values ranging from less than 10 to one to up to many thousands to one, and these high values are virtual proof of paternity.

The paternity index (PI) may be converted to a percentage relative chance of paternity (RCP) using the formula: RCP= PI/(PI+1). Thus a paternity index of 20:1 gives a relative chance of paternity of 95%; paternity indices above 100:1 give relative chances of paternity above 99%. When the index is higher than this further increases make little difference to the relative chance of paternity. Thus an index of 1000:1 gives a relative chance of paternity of 99·9% (virtual proof of paternity) and the figure is increased by only 0·09% if the index reaches 10 000:1.

In a small number of cases where the paternity index is less than 10:1 (relative chance of paternity less than 90%) additional tests like DNA fingerprinting are necessary. But usually traditional blood testing will settle the question.

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