to reduce pain and the misery that goes with it. At the conference Hinton was quoted: "We emerge deserving of little credit; we who are capable of ignoring the conditions which make muted people suffer. The dissatisfied dead cannot noise about the negligence they have experienced." Awareness is perhaps the most urgent need.

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Medical treatment of open-angle glaucoma

In the last decade much attention has been focused on finding the ideal drug for open-angle glaucoma. What we need is a non-toxic substance that will effectively lower the intraocular pressure over a long period without producing side effects. Propranolol is a most effective drug, but it is inconvenient for patients because it causes miosis and alters accommodation. Some patients are affected more than others, especially the younger ones with myopia and those with central lens opacities. Is there a satisfactory non-miotic drug? Adrenaline is the obvious answer, and it has been a useful drug for glaucoma for many years. But it has a limited effect, and unfortunately, used alone, its action is seldom sufficient to bring the glaucoma under control. Other catecholamines have been tried. Isoprenaline was effective, but it produced a tachycardia even when used topically and had to be abandoned. Salbutamol was equally effective but caused extreme hyperaemia and discomfort and could not be tolerated. Noradrenaline is less potent than adrenaline, which remains the best compromise in this group of drugs.

The next approach was to attempt to potentiate the action of adrenaline. Among the agents tried in various countries were 6-hydroxydopamine, protriptyline, and guanethidine. The combination of guanethidine with adrenaline provides a useful alternative to pilocarpine for patients with open-angle glaucoma. The solution is instilled only twice a day and has little effect on vision. About a third of the patients using this regimen suffer from hyperaemia, which may occasionally mean withdrawing the treatment, but most prefer a slightly red eye to the visual disturbances caused by pilocarpine. Tachyphylaxis develops only rarely, and many patients have been using this combination for over five years. The discovery that the adrenergic beta-blocking agents reduced intraocular pressure—with either systemic or topical administration—raised great hopes. Propranolol was the first compound investigated, and the early studies showed that when given by mouth it lowered intraocular pressure. Unfortunately the solution for topical application was unsuitable: it acted as a local anaesthetic and was also extremely irritating.

Other beta-blocking agents investigated included topical practolol, which, used in Holland, produced successful results for over a year; but it had to be withdrawn when side effects were reported. Topical atenolol gave promising results in initial studies. There was a profound fall in intraocular pressure, lasting six hours, after the early applications of the 4% solution. The long-term effect, however, has been disappointing. A gradual reduction in the effect is seen in about 75% of patients and eventually complete tachyphylaxis occurs about six months after starting treatment. Thus only 25% of the patients with open-angle glaucoma are suitable for treatment with this drug alone. Those patients who do not develop tachyphylaxis with atenolol report no discomfort or side effects: the pupil diameter remains unchanged and there is no hyperaemia. Timolol may prove a valuable alternative: it is more potent than atenolol and the incidence of tachyphylaxis appears to be lower. This drug is not yet available in Britain.

Systemically administered beta-blocking drugs are suitable for patients with both systemic and ocular hypertension. Propranolol has been used for several years for this group of patients. Tachyphylaxis is seen less often when the drugs are administered systemically.

The medical treatment of glaucoma is in a constant state of flux as new drugs are introduced and withdrawn; only time will tell which ones will find an established place. A hundred years after it was first used the place of pilocarpine is at last being seriously challenged.

Cannabis and the cardiovascular system

The effects of cannabis on the cardiovascular system are worthy of study for at least three reasons. Firstly, cannabis smoking has become so widespread that we should know what effects it has on patients with heart disease. Reefer smoke commonly contains nicotine, carbon monoxide, and tar as well as the active principles of cannabis. Cannabis depresses cardiac contractility in patients with angina pectoris; and even in the absence of heart disease long-term deleterious effects on the heart and blood vessels remain a possibility. Secondly, we need to know about any drug interactions that might occur when cannabis is taken with another drug. For example, in the presence of tetrahydrocannabinol (THC) atropine produces an appreciable pressor effect, and anticholinergic drugs or local anaesthetic containing adrenaline could dangerously potentiate a cannabis-induced tachycardia. Thirdly, among the many actions of cannabis, the hypotensive effect may conceivably have a clinical application—though