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Assaults on doctors

The popular stereotype of the doctor cultivated by the mass media is a composite of wisdom, equanimity—and invulnerability. The picture is complete only when framed in a white coat, and it may be this garment which, in some magical way, is thought to render its wearer immune from attack. In real life, however, medicine is a high-risk profession. Within the past two years in Britain a general practitioner has been killed and two consultants, one a psychiatrist and the other a general physician, gravely injured by mentally disturbed patients. In all three cases the assailants were paranoid schizophrenics who had harboured delusions—against specific doctors in two cases and against doctors in general in the third.

Doctors may be increasingly exposed to danger as a result of the more liberal discharge policies from mental hospitals; the lack of community care facilities after discharge; and a growing reluctance among social workers and psychiatrists to use compulsory orders to admit violent, or potentially violent, patients to hospital. Violence may occur symptomatically in virtually every variety of mental disorder. Sudden outbursts of unprovoked, unpredictable, and inexplicable violence may arise in catatonic schizophrenia. Conversely, in paranoid schizophrenia, the cold, calculated assault may be the result of years of growing resentment of some imaginary wrong. In mania assaults may be provoked by any attempt to prevent the patient doing whatever he feels called upon to do, no matter how fatuous or dangerous that may be.

Though far more a danger to themselves, victims of depression may occasionally turn on others. Hysterics in the process of acting-out may hurt themselves or those who attempt to restrain them. Those suffering from dementia are disinhibited and may become cantankerous and verbally, if not physically, aggressive. Lastly, the casual, callous violence of which the psychopath can be guilty was shown only too clearly in the murderous escape of two prisoners from Carstairs Hospital.[1 2]

By no means all the physical hazards stem from the mentally disordered. The brain is exquisitely sensitive to any alterations in the checks and balances that keep it finely tuned, and aggressive behaviour may result from toxic-confusional states such as the delirium seen in febrile illnesses; chemical intoxication, particularly by alcohol; drug intolerance, especially in old people whose sensitivity to even minor psychotropic drugs is well known; epilepsy; and brain damage, no matter how caused.

Of special importance nowadays is disordered behaviour due to abuse of drugs such as amphetamines, barbiturates, lysergic acid, and to a lesser extent, cocaine and the derivatives of opium. An associated hazard for general practitioners is the threat of assault, if not actual assault, by addicts should their demands for drugs or prescriptions not be met. The danger is heightened if it becomes known in the drug subculture that a doctor is a "soft touch."

Surprisingly little has been written by doctors for doctors on how to reduce the danger of assault in the day-to-day conduct of their practice. A report recently published by COHSE,[3] designed primarily for its members working in mental hospitals, contains a lot of practical advice of interest to doctors too. Some additional guidelines may be offered. In his surgery the doctor should face the door: there should be an alarm bell, discreetly hidden, but easily accessible. Help of some sort should always be readily available. In a threatening atmosphere the doctor should avoid any sudden movement which might be interpreted as an impending attack on the might-be assailant. If it does come to a show-down the doctor, with the aid of anyone to hand, is entitled to use minimum necessary restraints.

If time allows help to be summoned, the police have far-reaching powers. Under Section 40 of the Mental Health Act, 1959, they can return compulsorily detained patients who are absent without leave from mental hospitals. In addition, they have authority under common law and other statutory powers to prevent a breach of the peace and, if called upon, to give help where a mentally disordered patient may be deemed too dangerous to be at large. Social workers, particularly if they have been appointed mental welfare officers, have a vital role. Often they have personal knowledge of the patient concerned. Mental welfare officers are also authorised to make the application for the patient's compulsory admission to hospital in any case of urgent necessity.

Relative newcomers to the team of helpers are the community psychiatric nurses. They are trained mental nurses who liaise among the base mental hospital, the local authority, and the general practitioner. They too may be well acquainted with the disturbed patient, who may greet them as a friend and ally when all around seem strange and threatening.

It may be essential, and in all the circumstances it is legally permissible, to administer a sedative drug to calm a dangerous patient. The choice may seem wide, but in such circumstances

it may be best to rely on a drug such as chlorpromazine with an established reputation and familiar and predictable effects. The key to control is the adequacy of the initial dose or doses. Finally, the doctor should play it cool and, if at all possible, avoid going it alone. These precautions are axiomatic. There is little merit in posthumous awards for gallantry.

[1] *State Hospital, Carstairs: Report of Public Local Inquiry into Circumstances Surrounding the Escape of Two Patients on 30 November 1976 and into Security and other Arrangements at the Hospital.* Edinburgh, HMSO, 1977.

[2] *British Medical Journal*, 1977, **2**, 1374.

[3] Confederation of Health Service Employees, *The Management of Violent or Potentially Violent Patients. Report of a special working party.* Banstead, COHSE, 1977.

Ocular hypertension

Primary glaucoma remains an important cause of blindness in Britain: it accounts[1] for 7% of all new blind registrations and 10% in the age group 50-64. By far the most common type is chronic simple glaucoma—a degenerative disease of unknown aetiology in which increased intraocular pressure is assumed to result from progressive failure of the mechanism for aqueous outflow from the eye.[2] Population surveys have shown that this disease affects about 250 000 people in Britain.[3 4]

Chronic simple glaucoma is a silent disease, rarely producing symptoms until well advanced with extensive field loss in one eye at least. The diagnostic features are an intraocular pressure greater than 21 mm Hg, enlargement of the optic cup, and visual field loss. Because of its high prevalence and morbidity, much effort has been expended on discovering the natural history of the disease as well as identifying those who run the greatest risk of developing it.

Surveys[3 5 6] show that 8-10% of the population over 40 years have intraocular pressures persistently above 21 mm Hg without other signs of glaucoma—so-called ocular hypertension. Until recently all such cases were seen as "early" chronic simple glaucoma; in time, it was thought, enlarged optic cups—glaucomatous cupping—and field loss[7 8] would inevitably develop. Follow-up studies have shown, however, that this progression does not always occur; indeed some eyes become normotensive.[9] Furthermore, by no means every patient who develops chronic simple glaucoma can be shown to have had years of ocular hypertension.[10] Actuarial analysis shows that the number of people with ocular hypertension in the general population is far more than would be expected to develop obvious chronic simple glaucoma.[3] Even so, someone with ocular hypertension does seem to run a higher risk of developing chronic simple glaucoma than does a normotensive person, and the higher the intraocular pressure the greater the risk.[11 12]

When trying to decide which patients with ocular hypertension will, if untreated, develop glaucomatous cupping and field loss the ophthalmologist relies on several clinical signs. There are some characteristic changes that precede the development of readily detectable field loss. The optic cups, which are normally similar in shape[13–15] and have a diameter of less than 5/10 that of the optic disc,[15 16] gradually enlarge. This glaucomatous enlargement is typically along the vertical axis,[16–19] producing asymmetry between the optic cups of the two eyes,[20 21] and differs from the circular enlargement that occurs with age.[22 23] Serial fundus photography has shown progressive loss of the retinal nerve-fibre layer in patients with ocular hypertension who subsequently develop field loss.[24] Some people with ocular hypertension fare less well than those with normotensive eyes on colour vision testing[25] and visual acuity gratings,[26 27] which suggests that they may in fact have chronic simple glaucoma.

The diagnosis of ocular hypertension calls for an annual review of the intraocular pressure and the appearance of the optic discs, and testing of the visual fields. Long-term anti-glaucoma treatment is not without complications, however, and treatment should be reserved for patients with abnormal signs and those considered at special risk for other reasons.

Who are these high-risk patients? Firstly, those with very high intraocular pressures seem more likely than others to develop field loss.[11 12] Secondly, close relatives of patients with glaucoma have a tenfold increase in risk,[28 29] and the incidence of the disease is also higher in patients with myopia[30] and diabetes[31] than in the general population. Thirdly, several markers have been proposed for identifying patients (normotensive as well as those with ocular hypertension) who may be expected to develop chronic simple glaucoma.

In a small proportion of normal individuals[32 33] and most patients with chronic simple glaucoma[33 34] the intraocular pressure rises after they have been given topical steroids. This response is thought to be diagnostic of glaucoma or future glaucoma.[34] Normal intraocular pressures undergo diurnal variation,[35 36] closely following changes in plasma cortisol concentrations[37]; and this variation is increased in patients with chronic simple glaucoma, suggesting altered ocular sensitivity to plasma cortisol.[38] The plasma cortisol concentration is more easily suppressed in patients with glaucoma,[39 40] and has been suggested as a predictive test for glaucoma.[39] Moreover, patients with glaucoma have been reported by some[41–43] but not by others[44 45] to show an abnormally high incidence of steroid-induced inhibition of lymphocyte transformation. The relation between the "steroid response" and chronic simple glaucoma is, however, still in doubt; as yet no long-term study of steroid responders has shown large numbers progressing to chronic simple glaucoma.[46] Finally, the trabecular meshwork of eyes with steroid-induced glaucoma is quite different from that of eyes with chronic simple glaucoma.[47]

Two further screening tests have been described recently, based on the extent of the fall in intraocular pressure after topical adrenaline[48] and a possible link between glaucoma and HLA B12 and HLA B7.[49] Both associations require further evaluation before we can assess their usefulness for clinical screening.

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