Where should we train doctors in the future?

Less in hospitals, more in general practices

From the early nineteenth century until the late twentieth century it has seemed natural, indeed obvious, that medical students should receive their clinical education in teaching hospitals where medical skill and patients with serious illness were concentrated. This system is now under educational and financial threat. Professional and demographic changes and the action of market forces require a review of the fundamental assumptions of basic medical education.

Educationally, the threats can be divided into those that are already with us and those that are as yet only grim prospects. Dwindling local populations combined with the concentration of tertiary care facilities in teaching centres have seriously unbalanced an already highly unrepresentative case mix. The gradual loss of general physicians and surgeons from the staffs of teaching hospitals means that the teaching that students receive has become both more specialised¹ and more subject to chance. Specialists' interests bias the cases referred to them, which inevitably biases the teaching given to students attached to their firms. Luck usually plays a larger part in what students learn than does curricular planning. Other educational threats come from the near certainty that time and enthusiasm for teaching will be squeezed both by the service consequences of *Achieving a Balance*² and the absolute necessity for academic departments to boost their income from grants and their output of research. Finally, at a practical level, every pressure for shorter hospital stays and more intensive investigation, day care surgery, and community care reduces the opportunity for students to study illness by the patient's hospital bed.

At the same time the financial outlook is very uncertain. The future of the service increment for teaching and research still worries deans. The Department of Health's call for action to unravel the longstanding "knock for knock" arrangements between universities and health authorities may have become temporarily less insistent, but in the NHS of the 1990s managers are not going to be content to allow specialists to devote time to teaching without this time being somehow quantified and paid for. Even less can be expected if teaching hospitals opt for independent trust status.

What is the alternative to simply gritting our teeth and making the most of whatever resources are available? It is

crucial to remember that we are considering basic medical education—that which all medical graduates should experience before choosing and training for a specialty. Although it cannot be deep, it should be broad. It must not only introduce students to the specialties but also to medicine in all its guises, ensuring that students recognise that attitudes and skills, as well as knowledge, are essential to the education of an effective doctor.

The assumption of the past 200 years has been that only in hospitals does the requisite combination of teaching skills and patient morbidity come together. There are strong arguments, however, based on the intensity, breadth, and continuity of contact with patients, that generalists could play a much larger part than they do now.³ Patients need not be lost to the medical education system just because they are outside the hospital: simultaneous education in primary and secondary care should be our aim. Students, by being physically based in general practices for part of their time, could follow the progress of illness from the first report of symptoms through hospital care to resolution. Their education would be richer as a result. There are huge resources for teaching in the community if we can only find ways of tapping them. Developments are needed on several fronts. Hospital teachers and academic departments of general practice should get together to discuss the threats to education and what they could do about them. We need a group of GP teachers for whom undergraduate education is the main professional activity. We need to know more about the morbidity—and therefore teaching potential—of defined practice populations—and the acceptability to patients, students, and GP teachers of placing students in practices. The problems of medical education will not go away. Together, teachers in hospitals and general practice can overcome them.

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Managing violence in psychiatric hospitals

Empathy before drugs

Probably one in 10 psychiatric patients assaults staff, although this may be an underestimate.^{1,2} In psychiatric wards the typical patient who assaults staff is a young psychotic man who tends to remain in hospital longer than his non-violent counterpart. He is more likely to be held formally and to have a history of violence and previous admissions.^{3,4} Such patients may suffer from schizophrenia, substance misuse, or an organic brain syndrome. Hostility and poor control of impulses are the most consistent predictors of assault, although accurate prediction is possible in no more than a third of

cases.⁵ Very anxious, aroused, or paranoid patients are the most likely to express themselves in physical violence. Alcohol, stimulant drugs, and benzodiazepines contribute to disinhibition and the subsequent threat of violence. Warning signs are usually present: they include rapid breathing, clenched fists, loud talking or chanting, restless or repetitive movements, violent gestures, pacing, and gesticulating.⁶

Forensic units have the most disturbed patients but few assaults, reflecting high staffing ratios. Violence, when it does occur, is usually more serious and results in greater injury. In

special hospitals women patients are involved in three out of four incidents, and nursing staff are three times more likely to be assaulted than patients.^{7,8}

Staff should avoid being confronted by a violent patient when alone or in an enclosed space with the exit blocked. Ideally, each ward or reception area should have a discreet alarm. The best management is talking to the patient and encouraging the expression of aggressive feelings. The intention is to promote an atmosphere of empathy, thus gaining cooperation. Staff should use calm "body language"—for example, a relaxed posture with hands unclenched and an attentive expression. Threats should be taken seriously, staring eye contact avoided, and no attempt made to touch the patient. When possible, details of the history should be obtained in a sympathetic and unbiased manner to determine whether a psychiatric disorder (for example, psychosis or personality disorder) is present.

Some threatening and difficult patients may need seclusion. Increasing the level of supervision, however, allows most to be managed without this, thus avoiding excessive drug treatment. One study has reported that episodes of continuous observation lasting less than 72 hours helped two thirds of patients.⁹ Compulsory admission, lower social class, female gender, and a history of self harm and violence to property predicted those patients requiring continuous observation. After 72 hours alternative management procedures should be initiated, such as transfer to an intensive care unit, seclusion, or drugs.

Neuroleptic drugs are the best treatment when violence stems from psychotic conditions, though little agreement exists on the optimal drug. Indeed, violent behaviour (with heightened activity and excitement) increases in some patients given high doses of haloperidol (60 mg a day).¹⁰ Fortunately, most patients respond to 50 mg a day (or less) or a similar drug such as droperidol, which is more sedating, acts more quickly, but does not have such long lasting effects.

Akathisia (distressing motor restlessness), a side effect of neuroleptics, aggravates the clinical condition (as do extrapyramidal side effects) and has been implicated in violent behaviour in schizophrenic patients. It is frequently resistant to prophylactic antiparkinsonian treatment. Chlorpromazine is highly sedative, unlike haloperidol, and may suppress violent behaviour in the same way as the benzodiazepines. Both chlorpromazine and the benzodiazepines may, however, have a paradoxical effect through disinhibition of the nervous system.¹⁰ Chlorpromazine 50-200 mg can be given intramuscularly; this dose may need repeating up to a total daily

dose of 1 g. The injections are sometimes painful and in high doses are likely to cause hypotension and tachycardia. Zuclopenthixol acetate may be useful in some patients: one study reported that a single injection (50-150 mg) had an antipsychotic effect lasting three days.¹¹ Sedation and calming of mood and motor behaviour occur rapidly (within 15-90 minutes of injection) and are maximum after eight to 12 hours. Side effects are rare and mild.

Benzodiazepines, particularly diazepam, and barbiturates (intravenous amylobarbitone sodium) are sedating but uncommonly cause disinhibition and paradoxical aggression, and also respiratory depression. Conversely, when aggressive patients are treated with a combination of lorazepam and a neuroleptic agent the neuroleptic agent may not need repeating so often. Propranolol (and other β blockers), either alone or in combination with neuroleptic drugs, diminishes aggressive behaviour, but daily doses of 200-800 mg or more for four to eight weeks may be necessary, and bradycardia or hypotension sometimes limits treatment. Carbamazepine may be useful for patients with epilepsy and associated "irritability." It is synergistic with lithium in treating explosive violence in patients who are refractory to more conventional treatments,¹² but slightly higher blood concentrations may be required than are needed for typical seizures.

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The familial transmission of alcoholism

Imitation and inheritance

Alcoholism is known to run in families—the relatives of problem drinkers have a greatly increased risk of the disorder—but the ways in which this has been explained have changed over the years. For three decades after the second world war transmission was thought to be by imitation rather than inheritance. Since 1973, however, when Goodwin *et al* suggested that alcoholism was transmitted genetically, the alcohol journals have burgeoned with genetic research.

Goodwin *et al* compared the risks of alcoholism in the sons of problem drinkers who had been adopted by other families

with the risks in control adoptees; those who had received their genes from an alcoholic parent had a fourfold increased risk of alcoholism themselves, even though they had been brought up in a normal family.¹ Other adoption studies, from Iowa² and Sweden,³ confirmed the genetic effect. Two subsequent approaches have attracted particular attention. Firstly, Cloninger used data from the Swedish adoption study to propose two subtypes of alcoholism.⁴ Type I, a mild form, occurred in both men and women, had a late onset, and was influenced by both genes and the environment. On the other