The special hospitals

Should be closed

What should be done about the special hospitals? The report of the inquiry into Ashworth Hospital, published this week, raise serious and urgent questions about the way in which we care for our most vulnerable, disturbed, and difficult to manage psychiatric patients in the future.

The committee of inquiry, chaired by Sir Louis Blom-Cooper QC, was formed in April last year after a television documentary on Channel 4 alleged improper care and treatment (mainly physical ill treatment) at Ashworth Hospital, and its thorough and detailed report confirms these allegations in four cases investigated in depth. A wider inquiry into the milieu within which these serious incidents occurred reveals a shameful failure to provide a modern therapeutic environment for patients, supported by multi-professional services of at least adequate standard, and led by a firm management structure. The report refers to “an uncaring and demeaning attitude towards patients,” some harassment and physical bullying, a low standard of care, and poor standards of therapeutic practice—which were not confined to nursing staff. A lack of clinical leadership and an absence of firm hospital management left “a power vacuum,” all too readily filled by the main trade union, the Prison Officers’ Association. According to the report, the union continues to give priority to security, control, and discipline, which take precedence over a climate of treatment (an observation framed in almost identical terms with that made by the Boynton inquiry into similar allegations at Rampton Hospital over a decade ago).

During the 15 years before the establishment of the Mental Health Act Commission in 1983 there were a series of major scandals concerning neglect, inadequate care, and ill treatment in psychiatric hospitals, with at least 10 national inquiries and many local ones. Once the commission had the power to inspect hospitals and protect the rights of patients such inquiries were expected no longer to be needed. Despite its best efforts, however, the commission has clearly been ineffective in uncovering a myriad of problems in Ashworth Hospital and preventing yet another painful and expensive investigation, even though its biennial reports have expressed concern and the need to probe more deeply.

The impotence of the Mental Health Act Commission is shared by the Special Hospitals Service Authority, which was established by the Department of Health in 1989 to take over the management of the four special hospitals—Moss Side and Park Lane (now amalgamated as Ashworth), Broadmoor, and Rampton—from the Special Hospitals Service Board, which previously administered the hospitals on behalf of the secretary of state. The new authority was given the objectives of raising standards of performance and promoting closer links, and ultimately integration, with the rest of the NHS.

The three special hospitals have not been able to rid themselves of an institutionalised culture of geographical, therapeutic, and professional isolation, which can be traced to their origins within the penal system until 1946, when the transfer of their management to the Ministry of Health started. Nursing staff have continued to join the Prison Officers’ Association, and, although there have been many notable advances, the continuation of these large and unwieldy institutions into the 1990s, when most large mental hospitals have closed, perpetuates anachronistic attitudes and makes the altruistic aim of transforming them into “centres of excellence” difficult, if not impossible.

Although the improvements that have been made at Rampton since the Boynton report7 and at Broadmoor since the critical report of the Health Advisory Service of 19888 must not be underestimated or undervalued, it would be inappropriate to sustain these three hospitals in the future. A radical change in policy is needed to provide for the mentally disordered with dangerous, violent, or criminal propensities (of whom about 20% are not offenders). A more efficient use of the Special Hospitals Service Authority’s resources for its 1700 patients (£85m revenue and £20m capital allocated in 1991-2) is required.

Most of the patients should be cared for in new local high security units, perhaps one shared by two health regions, of about 100-150 beds; these should be linked to local regional secure units and psychiatric services, close to the patients’ friends and families, and geographically placed to allow a smooth continuity of care into the community. The prevailing culture of the old institutions must be replaced by an emphasis on care, treatment, stimulation, and rehabilitation. Security should be regarded as integral to treatment rather than the main objective. Proper value should be given to civil rights, to abandoning oppressive methods of control (including the excessive use of seclusion), and to recognising the patient’s autonomy. The regional secure units have shown...
Unawareness of hypoglycaemia and human insulin

Human insulin almost exonerated

A fall in plasma glucose concentration below normal usually elicits a characteristic hierarchy of responses. At a concentration of about 3-8 mmol/l the secretion of counterregulatory hormones increases and activation of the autonomic nervous system occurs. Should the decrease in concentration persist or progress, additional release of neurohormones produces autonomic warning symptoms (sweating, anxiety, palpitations, hunger, and tremor), which may prompt the affected person to take protective action (that is, to ingest carbohydrate). These warning symptoms generally occur at a plasma glucose concentration of 3-4 mmol/l, which is about 0-5 mmol/l above that at which neuroglycopenic symptoms and cognitive dysfunction begin.

The failure of autonomic warning signals to occur before neuroglycopenia develops is commonly referred to as hypoglycaemia unawareness. First described shortly after the introduction of animal insulin, it has also been noted in patients with insulinoma, spinal cord section, ganglionic blockade, and autonomic neuropathy. In insulin dependent diabetic patients it is associated with a long duration of diabetes, frequent episodes of severe hypoglycaemia, and strict glycaemic control, though not necessarily with autonomic neuropathy. Patients with insulin dependent diabetes require more severe hypoglycaemia to initiate secretion of counterregulatory hormone and reduce secretion of adrenaline and noradrenaline. Episodes of hypoglycaemia may induce these changes; aside from this, other causes remain speculative.

One speculation, which has received considerable attention in the lay and scientific press, is that human insulin may diminish patients' awareness of hypoglycaemia, thereby increasing the risk of severe hypoglycaemia. This issue was reviewed in 1987 and again in 1989; both times no conclusive evidence was found. In fact, this was the conclusion of an expert panel of the United States Food and Drug Administration after a public hearing in 1989. Two articles in this issue of the journal again review this question and propose the need for a large multicentre randomised trial (p 355, p 351). In 1989, on the basis of available evidence, most practitioners, professional diabetes organisations, and government agencies did not consider human insulin to be an important threat to health. Has there been additional incriminating evidence since 1989 to warrant undertaking a large multicentre trial?

Such a study would be justified if the hypothesis of harm was plausible or if the outcome could improve the safety and efficacy of treatment. Regarding plausibility, nothing has changed. As Williams and Patrick point out, there is still no established physiological basis for an intrinsic effect of human insulin to alter autonomic activation in response to hypoglycaemia and to reduce awareness of hypoglycaemia. This would presumably have to occur as a result of insulin acting on the brain or the endothelial cells of the blood-brain barrier. Because human and porcine insulin bind to insulin receptors identically differential access to receptors must be postulated. But there is no evidence for such an effect. The subtle difference in subcutaneous absorption between human and porcine insulins is trivial when compared with the intrasubject variations in subcutaneous absorption of both insulins. Thus how human insulin could intrinsically reduce autonomic activation in response to hypoglycaemia remains a mystery.

Furthermore, the studies cited by Egger et al and others not cited indicate that no new evidence exists that human insulin reduces plasma catecholamine responses to hypoglycaemia. In fact, the opposite is true. All nine studies since 1989 that compared porcine and human insulin found no significant difference in plasma catecholamine responses or autonomic symptoms. Egger et al suggest that the small number of subjects studied may have limited the power to detect a difference. Maran et al, however, studied 17 patients with insulin dependent diabetes in a double blind crossover trial, using the sensitive stepwise hypoglycaemic clamp technique, and found no difference. Such a study should have had the power to detect a clinically important difference if one existed. In summary, there remains no known physiological basis by which human insulin can intrinsically affect autonomic activation in response to hypoglycaemia, and direct comparisons of human and porcine insulins under carefully controlled conditions persistently fail to show that human insulin reduces plasma catecholamine responses to hypoglycaemia. One can therefore seriously question the plausibility of the hypothesis for the proposed multicentre study.

This conclusion is supported by postmarketing surveys and many other studies, which have failed to show an increased frequency of severe hypoglycaemia in patients treated with human insulin (as pointed out by both Williams and Patrick).