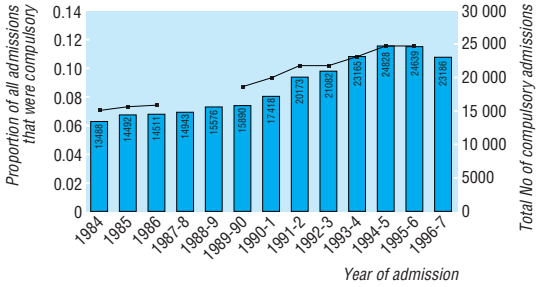

Bars represent the total number of compulsory psychiatric admissions to NHS facilities and the line represents the proportion of all admissions that were compulsory in England, 1984-96. Data on compulsory admissions not available for 1987-9

For 1989-96, when only one source of data was used for the denominator, the trend remained constant.

What explanations are there for the increase in compulsory admissions? Firstly, these changes may be due to alterations in the presentation of patients with psychiatric disorders. For example, there is some evidence that a higher proportion of psychiatric patients misuse drugs and alcohol, and this may lead to more florid presentations of psychotic illness.[2] Secondly, changes in the availability of beds during this period may have increased the threshold for admission and decreased the threshold for discharge. Between 1982 and 1992, approximately 43 000 fewer psychiatric hospital beds were available,[3] and in inner city areas bed occupancy remains above 100% much of the time.[4] The public's fear of violence by mentally ill patients and pressures to keep patients in hospital until it is "safe" to discharge them put further strain on the availability of beds. Delays in admission and treatment caused by bed shortages may mean that patients' illnesses are becoming more severe and that compulsory treatment is being initiated in cases in which informal admissions would previously have been possible.

These results have implications for resources in terms of costs and staffing. Compulsory admissions are more time consuming since they generally require that patients be assessed by two doctors and a social worker. Disturbed patients also require more intensive nursing and supervision. Patients admitted under the act have the right to appeal, and mental health tribunals are time consuming and costly. We suggest that the move to community care may have led to a paradoxical and unexpected increase in the use of coercion in the treatment of patients with mental illnesses.

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Drug points

Aseptic meningitis after treatment with amoxicillin

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The development of aseptic meningitis has been associated with various drugs—for example, non-steroidal anti-inflammatory drugs, ranitidine, carbamazepine, vaccines against hepatitis B and mumps, immunoglobulins, co-trimoxazole, and penicillin.[1–5] We report a case of aseptic meningitis after treatment with amoxicillin.

A 76 year old woman was admitted to our hospital with fever, headache, and neck stiffness. Five days before admission she had had a pretibial wound treated with amoxicillin-clavulanic acid. Long term treatment with aspirin, enalapril, and levothyroxine (thyroxine) had not been changed in the previous month. Two days before admission she had developed fever, headache, and neck ache.

On admission her general condition was poor, but findings on physical examination were normal except for neck stiffness. All laboratory findings were within the normal range. Cerebrospinal fluid showed pleocytosis with 63 cells (62 monocytes) and a slightly raised protein concentration of 0.47 g/l (0.15-0.45 g/l). No micro-organisms were found. She recovered with treatment of symptoms.

From her history we knew of two similar episodes in 1992 and 1995. Twelve and 6 days respectively after the initiation of antibiotic treatment with amoxicillin (with and without clavulanic acid), she had been admitted to our hospital with the same symptoms of fever, headache, and neck stiffness. Cerebrospinal fluid had been examined during the first admission and also showed pleocytosis with 40 cells (38 monocytes) without an increase in protein concentration. No bacterial micro-organisms or serological signs of neurotropic viral infections had been found.

On the basis of these three confirmed episodes of meningitis after recurrent exposure to amoxicillin, with and without clavulanic acid, with repetitive negative testing for viral, bacterial, and mycobacterial micro-organisms, we diagnosed aseptic meningitis induced by amoxicillin. To our knowledge, this is the first well documented publication of such a severe side effect of a commonly used antibiotic.

The exact mechanism for the development of aseptic meningitis induced by drugs is not known, although hypersensitivity reactions and immunological mechanisms have been suggested.[5]

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