

intuition that a unit with only 72% occupancy and an average length of stay of 21 days could not possibly have an average waiting time of 84 days. I regret that the results given by him in the second example and in the table are also incorrect.

There are two other points worth making. The first is that despite Dr Marjot's assertion the expediency of borrowing beds between firms does indeed reduce waiting times provided the overall occupancy is less than 100%. To take an extreme example, consider two units, each with only one bed, and each with a 50% occupancy. If you want to admit a patient to one unit the probability of finding the bed full is 0.5. However, if you can admit the patient to either bed then the probability of finding both beds full is only about 0.25 (not exactly because the two situations are not truly independent). The more beds available, the more they can absorb the fluctuations in admission demand which produce queues.

The second point concerns the limitations of mathematical models in general. In reality there is no clear distinction between patients who need admission and those who do not, perhaps especially in psychiatry. Every trainee knows that they are more likely to admit patients when there are plenty of empty beds than when there are only one or two. This kind of feedback loop is not really possible to incorporate adequately in a model. Patients in a queue do not remain there indefinitely. Some get better, some go elsewhere, some die. The length of time spent waiting may influence the length of admission, for example in surgery for malignancy. These considerations and others should mean that we take with a pinch of salt any mathematical model which purports to predict reality, especially if its predictions fail to match with common sense.

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REFERENCES

- ¹SINGH, J. (1968) *Operations Research*. Harmondsworth: Penguin
²KAUFMAN, A. (1963) *Methods and Models of Operations Research*. Hemel Hempstead: Prentice Hall.

DEAR SIRS

I am sorry that Dr Curtis has rendered my (very amateur) maths invalid. I will try and resolve the blow to my ego!

Nonetheless I was delighted that he could propose a model to allow us to see more clearly the effects of demand on our services and the resulting queues. He has given us a more valid tool.

An example I gave, and which Dr Curtis has reworked, was that average bed occupancy would be 82%, i.e. 23 beds occupied on average out of 28. Yet managements can and do argue that you should therefore cut your service by five beds. I think Dr Curtis will agree that this would make a great difference to queues and admissions.

I agree that trainees (and consultants) are more likely to admit when there are plenty of beds. If we cannot incorporate this kind of feed-back into models, we are in

trouble. It is a subject that needs further study in its own right.

I also share the opinion that we must take with very large pinches of salt any mathematical model that purports to predict reality (including rating scales and double blind controlled trials).

However my errors do not invalidate the need for more objective ways of discussing our resource needs with management. Rationing health care can be done in three ways. The first is by making the patient pay at the point of delivery of the service, out of income or capital which may be anticipated expenditure if insurance is used. The second way is to determine your use to the community, whether this be by the value of your survival or the cost of keeping you alive in any given state of health. Quality of life units and casualty triage are examples. Lastly you can ration by queue which is the way favoured by the NHS at present.

It is necessary for us to understand the mechanics of queueing in order to have rational services in the NHS and so I took tentative steps towards this end.

With Körner data sets and computerised management information systems about to run amok in the NHS it behoves us to get to grips with the theory and practice of these systems, including their very real limitations.

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DEAR SIRS

Dr Marjot, in his letter (*Bulletin*, December 1987) is quite correct to emphasise the point made by Prof Priest (*Bulletin*, November 1986) that in calculating the number of beds a unit needs, it is important to remember that 100% occupancy is not compatible with the functions of an acute ward. However, it is a shame that, like the recent College Working Party on bed norms, his study of the literature seems to have overlooked the contribution of by far the most original theoretician in this area, the late J. A. Baldwin.

In a paper published in 1963 Baldwin¹ noted the importance of the issues that Marjot discussed but he went on to make a further important point which Marjot seems to have missed. The overall number of beds in a unit influences the proportion of beds which need to be vacant to buffer normal fluctuations in the admission rate.

This is pretty obvious intuitively but it can be demonstrated by reference to poisson distributions. If a unit of 30 beds admits on average three patients a day, then in order to reduce the likelihood of having to turn away a patient on any single day to below 1%, eight empty beds will be required (26.7% of the total). By contrast a unit of 150 beds admitting, on average, 15 patients per day can achieve the same level of confidence in its capacity to admit as required by freeing only 25 beds (16.7%).

Unfortunately the language in which these issues are usually discussed emphasises the physical facilities (the bed). The staff implications are in practice likely to be more tricky. The point is that if in-patient psychiatric practice is to move to smaller units, it becomes much more important

to develop flexible arrangements for availability of nursing staff. This, with its attendant problems in terms of forming a cohesive staff group, is the only way to avoid the dangers of on the one hand generally excessive levels and on the other occasional dangerous inadequacies.

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REFERENCE

- ¹BALDWIN, J. A. (1963) A critique of the use of patient movement studies in the planning of mental health services. *Scottish Medical Journal*, 8, 227–233.

Community Treatment Orders

A Discussion Document of the Royal College of Psychiatrists

DEAR SIRS

It would appear that after an excellent description of the need for a compulsory Treatment Order in the Community, this document under paragraph 6, Procedures to Follow if Patients continue to Refuse Treatment, in the end concludes that compulsory treatment can only be given voluntarily; thus the order, with the back-up threat of rehospitalisation, becomes no more than blackmail to comply. This, however, seems to be because of poor use of words "... most patients will then agree to treatment. However, some will not and it is not proposed that the patient should be actually given medication compulsorily outside the hospital setting ... in the case of refusal ... admission to hospital is appropriate".

The issue in this paragraph would have been clearer if, instead of "not agree", the document had used "resist". What it is clearly trying to avoid is the inculcation of the use of what used to be called "a show of force" in the community: hence the suggestion that the patient, under such circumstances, be returned to hospital, where, presumably, the treatment would be forced if necessary.

This paragraph should then make it clearer that the Compulsory Treatment Order in the Community advocated in the rest of the document does mean compulsion and should be insisted on to the point at which resistance could only be met by force: at this point alone would readmission to hospital be considered.

As luck would have it, in my experience the schizophrenics who most need the compulsory treatment to avoid self-defeating relapse in the community not only refuse it if they possess the power, even against their own good estate, but, once they know compulsion exists and can lead to sanctions, comply readily, even to the point of regular visits to hospital for their depot injections.

I hope, then, the College will make clearer its position by strengthening the wording of paragraph 6 along the lines I have suggested.

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Judge Schreber's nervous illness

DEAR SIRS

In 1986 Dr Stanley¹ re-examined Judge Schreber's nervous illness in the *Bulletin*. His study was based on the English translation² of Schreber's autobiography, undoubtedly the most famous ever published. This is partly due to Freud using it as a starting point for his theory of paranoid psychosis. In addition though, as Baumeier³ wrote, "...the excellent presentation of his psychosis, the admirable objectivity of the description, and the even artistic imagination of his delusion make (it) ... a classical book which after 50 years (A. B.: and even after 85 years) has lost nothing of its attraction".

Stanley¹ ends his article by stating that the translators "...tried to discover the eventual outcome (of Schreber's illness) but were only able to establish that Schreber died in 1911 (and that) there is no mention of a post-mortem examination which Schreber said would provide 'stringent proof' that he suffered from a physical disease of the nervous system".

To provide that proof without a post-mortem is what Stanley¹ tries to accomplish. By an analysis of Schreber's writings, and by interpreting it against the background of relevant literature, the author suggests Schreber might have suffered from temporal lobe epilepsy and damages to other parts of his brain caused by encephalitis lethargica.

Having published the first autobiography of an African psychotic patient under the subtitle *A Schreber Case from Cameroon*,⁴ I had come across more recently published literature on Judge Schreber's case and I feel Stanley's interesting article requires a supplementation.

Macalpine & Hunter² mention briefly a first paper by Baumeier⁵ in which he reports on "a further psychotic breakdown in 1907 which lasted to his death in 1911" but they had not been able to verify it. In the year of MacAlpine & Hunter's publication Baumeier³ reported in a second, detailed paper how he found Schreber's original case notes of the Mental Hospital Leipzig-Dösen where Schreber was treated as an in-patient from 27 November 1907 until his death on 14 April 1911. The case notes reprinted in the paper include excerpts, some very extensive or even copies, of the case notes of 11 previous periods of Schreber's hospitalisation. Most relevant in the present context is the fact that the case notes also include, as Baumeier³ states, "... a very detailed post-mortem protocol" of which the summary (pathologisch-anatomische Diagnose) is reprinted as follows (translation into English of German terms by A. B.): 'Pleuritis exsudativa chronica. Pyothorax sinister. Atrophy of the left lung. Atelectasis of the left upper pulmonary lobe. Pericarditis fibrinosa acuta—Myode-generatio.—Sclerosis of the coronary arteries. Multiple haemorrhages into the pons cerebri".

Considering the high standard of brain pathology in the mental hospitals of that period it is justified to assume the post-mortem would have discovered any relics of brain disease if they had existed, especially signs of chronic, subacute, or previous encephalitis of any type.

Taking into account further that Dr Baumeier, whom I