MATTERS ARISING

Predicting the outcome of acute stroke

The multivariate models examined by Gladman, Harwood and Barer predicted death within three months of a stroke with an accuracy of only 50–75%.¹ It should be emphasised, however, that these models were not designed to predict mortality at three months; the Uppsala and Belfast models were designed to predict mortality at one and six months respectively, and the Guy's Hospital Prognostic Score was designed to predict death or functional dependence at two months. We have used data from an unselected cohort of patients registered with the Oxfordshire Community Stroke Project (OCSP)² to examine the utility of the Guy's score in predicting severe functional dependence or death at one and six months after a first stroke. For this analysis a modified Rankin grade³ of 4 or 5 was taken to indicate severe functional dependence.

When applied to the 165 patients registered in the final year of the OCSP, when the relevant data items were collected, the Guy's score predicted severe functional dependence or death one month after the stroke with a specificity of 99% and a sensitivity of 34% (predictive accuracy 77%). A modified version of the score which omitted one clinical feature, loss of consciousness at onset, but could be tested on the whole cohort of 675 patients, performed with a similar accuracy (table 1). With the sensitivity of the modified score equal to that of impaired conscious level (unconscious or drowsy at 24 hours), the likelihood ratio of the modified score (8.0) exceeded that of impaired conscious level (5.5), suggesting that when used to predict both functional dependence and death, the Guy's score did provide additional prognostic information.

The clinical usefulness of the Guy's score may, however, be limited. The calculation was laborious and the score required the identification of higher cerebral dysfunction which was difficult in acute stroke patients. A single clinical feature, such as, impaired conscious level might therefore be preferred. In this analysis urinary incontinence

Table 2 Predictors of death or dependency six months after stroke.

	Sensitivity	Specificity	Positive PV	Accuracy	Likelihood ratio
Drowsy/unconscious on Day 1	55%	81%	77%	67%	2.9
Incontinent of urine on Day 1	83%	65%	74%	75%	2.4
Drowsy/unconscious and incontinent on Day 1	52%	86%	81%	68%	3.7

(or the use of a urinary catheter), occurring between the time of the stroke and the examination by the study neurologist (median 4 days), was the most accurate predictor of a poor outcome (table).

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Gladman et al reply:

We agree with the comments of Drs Burn and Sandercock on our paper. The diversity of clinical measures required by the models we examined forced us to modify the predicted outcomes in some cases, to make comparisons. This did not significantly affect our overall conclusions and the Oxfordshire Community Stroke Project (OCSP) results appear to confirm this. It is not surprising that the Guy's score was slightly better at predicting bad outcome (death or dependency) than conscious level alone as the latter variable forms part of the Guy's model. As Burn and Sandercock point out, the extra effort in collecting the other data needed for the model and calculating the score is probably not justified by the practical gain.

The OCSP data also confirm, once again, the pre-eminence of continence as a predictor of functional outcome. If any addi-

tional information is required to modify the over-pessimistic forecasts of impaired conscious level alone, continence is the obvious choice. In our study we did not routinely collect information on continence on day 1 but we are able to test our proposal on an independent set of nearly 400 hospitalised stroke patients in whom this information was available.

Table 2 shows the accuracy of various predictors of death or dependency at six months (54% of the whole patient group). Thirteen per cent of drowsy patients were continent on day 1 and these were subtracted from the "bad prognosis" group to form a combined predictor (impaired conscious level and incontinent) with a likelihood ratio of 3.7 compared with 2.9 for impaired conscious level alone, and 2.4 for incontinence alone. A more elaborate combined scale involving different degrees of impaired consciousness and incontinence could also be constructed and might further improve predictive accuracy.

The practical value of all these predictive models and variables depends on the situation. Prognostic stratification is certainly important for clinical trials (and it may be impractical to use continence as a guide when very early medical intervention is required) and a simple system of "triage" may help in the efficient organisation of stroke services. We still maintain, however, that the modest gains in predictive accuracy provided by multivariate models are of negligible value in guiding the management of individual patients.

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Hypergraphia and brain damage

We wish to comment on the paper by Imamura et al¹ on hypergraphia and brain damage, to show that hypergraphia can also be an uncommon symptom of a dementia of frontal type.

A 60 year old policeman with a positive family history for dementia came to our attention in 1991 with a severe cognitive impairment. He first had difficulty in drawing schemes of car accidents nine years previously, and this was soon followed by personality and behaviour disturbances such as disinhibition, aggressive behaviour, impulsivity, emotional lability. The following few years he developed word finding problems, gluttony, ritualistic behaviour, hoarding of objects, decreased speech output, verbal stereotypes, paraphasias and mild memory loss. In 1987 the patient could still drive his own car without getting lost and the other main activities of daily living were only mildly affected.

In 1986 he started compulsively writing his own signature filling some 1500 sheets

Table 1 The positive predictive value and predictive accuracy of the Modified Guy's Hospital Prognostic Score and various single clinical features in predicting death/severe functional dependence at one and six months after a first stroke.

	Positive Predict Value		
	Death 30 days	Death/Functional Dependence 1 month	Death/Functional Dependence 6 months
Modified Guy's Score*	51%	85%	88%
Unconscious	75%	93%	92%
Unconscious/Drowsy	50%	79%	73%
Incontinent/Catheterised	47%	95%	85%
	Predictive Accuracy		
	Death 30 days	Death/Functional Dependence 1 month	Death/Functional Dependence 6 months
Modified Guy's Score*	81%	77%	81%
Unconscious	86%	59%	69%
Unconscious/Drowsy	81%	75%	74%
Incontinent/Catheterised	85%	88%	83%

*Cut off score: –2.5.

Figure Three examples of the patient's writing production, arranged in chronological order from top (at the beginning of the symptom), centre (about six months after), to bottom (about one and a half years after). Black marks outline right and left page margins.

of paper in about one and a half years. He could write for hours without getting tired. At the beginning of this symptom he could spontaneously write meaningful sentences, with a neat handwriting. His handwriting, which was inattentive to the left, became progressively careless and increasingly unreadable over time (fig), until his writing finally stopped late in 1987, about one and half years after the beginning of the symptom.

Brain CT scan at the time of admission showed moderate cortical and subcortical atrophy without anterior-posterior or right-left differential involvement, ruling out Pick's disease.² An EEG showed mainly frontal slow waves. This case, atypical for a dementia of the Alzheimer type, suggests instead a diagnosis of dementia of frontal type for the early personality and behavioural changes with a relative sparing of memory, topographic orientation, and function.³

Imamura *et al*'s patient was an 80 year old man with a metastatic brain tumour confined to the right hemisphere;¹ his hypergraphia was similar to the one described by Yamadori in stroke patients.⁴ Hypergraphia has not been reported in dementia of frontal type. In our patient it resembles Yamadori type hypergraphia^{1,4} as it is semi-automatic and inattentive to the left, has a poor communicative value, and the patient was totally indifferent to his writing production. As opposed to Yamadori type hypergraphia, however, hypergraphia in our patient was highly stereotyped, perseverative and, at least in the early stages, spatially well organised. These features suggest a frontal component in its pathogenesis.

Our case shows that hypergraphia may be an uncommon compulsive symptom of a frontal type dementia.

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Polyglucosan bodies are not an unusual finding in temporal lobe epilepsy

A recent short report excited my interest. The report described the massive occurrence of polyglucosan bodies (PBs) in a surgically-resected temporal lobe.¹ However, I am afraid that I have to disagree with the authors' contention that the occurrence of PBs is "very unusual in neuropathological examinations of epileptogenic foci".

Of the 40 temporal lobes resected for temporal lobe epilepsy which I recently reviewed, 15 contained corpora amylacea (PBs) in numbers far in excess of what would be expected for the patient's age and the anatomical site. In half of these the PBs were associated with mesial temporal (Ammon's horn) sclerosis and in the remainder they apparently constituted the sole abnormality.² Over half of the Liverpool cases with excess PBs also had a history of febrile convulsions in childhood, although not all with such a history manifested mesial temporal sclerosis. Although they claim that "unusually large numbers of PBs were the only significant abnormality", the fact that there was "severe neuronal loss in the pyramidal layer of the Ammon's horn" indicates that the case of Loiseau *et al* also demonstrated mesial temporal sclerosis.

The association with febrile convulsions and mesial temporal sclerosis suggests that the PBs are a result of either the epileptic

activity, or the original process which damaged the temporal lobe, rather than "the origin of an epileptic focus".

The authors do, however, correctly highlight the sparsity of previous references to PBs in epileptic temporal lobes—they are not mentioned in Bruton's detailed study of 249 cases,³ and only one of the 81 specimens studied by Jackson *et al* was reported as having unusually large numbers of white matter corpora amylacea.⁴ It is highly unlikely that the sparsity of reports reflects the rarity of epileptic temporal lobes containing excessive numbers of PBs. It is much more probable that the PBs have previously been overlooked by pathologists, either because their attention has been directed to more obvious pathology within the temporal lobe, or because the numbers of PBs have not been appreciated as excessive in relation to the patients' young ages (34 in Loiseau's case and a mean age of 24.6 for the Liverpool cases), or because they have been poorly stained (PBs stain intensely with Ehrlich's haematoxylin, which is routinely used in Liverpool, but weakly with Harris' and Meyer's haematoxylin).

If the interesting report by Loiseau *et al* stimulates pathologists to scrutinise more carefully resected temporal lobes, then it will have served a very useful purpose.

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Loiseau *et al* reply:

We were very interested in Dr MacKenzie's comments, and agree with his technical remarks concerning the use of Ehrlich's haematoxylin to stain PB. We were unaware of his data because our manuscript was submitted to the journal at the time that his abstract was published.

We maintain that the occurrence of PBs is unusual in neuropathological examinations of epileptogenic foci. It is hard to believe that PBs were not seen in so many temporal lobes examined by so many pathologists. We have carefully examined the largest series of pathological examinations of cortectomies throughout the available literature (cited in Bruton's monograph) and we found no evidence of occurrence of excessive amounts of PBs. Another pathological series of cortectomies was recently published by Swartz *et al*.¹ Abnormal amounts of PBs in temporal lobe were only found in three out of 37 cases.

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