	Height (m)	Body-mass index	Waist-hip ratio	Waist-bust ratio	Bust-hip ratio
Fashion models	1·77 (0·00)	17·57 (0·26)	0·71 (0·02)	0·72 (0·02)	0·99 (0·00)
Glamour models	1·69 (0·00)	18·09 (0·07)	0·68 (0·00)	0·66 (0·00)	1·03 (0·00)
Anorexic women	1·65 (0·01)	14·72 (0·36)	0·76 (0·01)	0·78 (0·01)	0·96 (0·00)
Bulimic women	1·65 (0·01)	23·66 (1·05)	0·77 (0·01)	0·83 (0·01)	0·93 (0·01)
Normal women	1·66 (0·00)	21·86 (0·22)	0·74 (0·00)	0·80 (0·00)	0·92 (0·00)

All values are mean (SE).

Biometric characteristics of women

two smaller samples of 30 anorexic women and 30 bulimic women recruited from Newcastle City Health's Eating Disorder Service.

For each woman we calculated the body-mass index, the waist-hip ratio, the waist-bust ratio, and the bust-hip ratio. These values give a summary of variation in body shape. Behavioural studies suggest that there is an optimum waist-hip ratio for female attractiveness,[4] men are supposed to find a waist-hip ratio of 0·7 to be most attractive; this ratio corresponds to a fat distribution which gives optimum fertility.[5]

Fashion models were significantly taller than all the other groups of women, on average 11 cm taller than normal women. Both fashion models and glamour models were rated significantly underweight on the basis of body-mass index, but were consistently heavier than anorexic women. Fashion models and glamour models had a waist-hip ratio close to the optimum of 0·7, and tended to have an hour-glass figure, as shown by the bust-hip ratio. Indeed, fashion and glamour models had similar measurements, although glamour models are usually regarded as more curvaceous than fashion models. The key difference may be height. A shorter hour-glass figure will appear more curvaceous. The take-home message from this study is that supermodels are both tall and curvaceous, and that dieting will not make you look like a *Vogue* covergirl.

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1 Frean A, Bradberry G. Vogue model too thin for Omega. *The Times* 1996; **May 31:** p 1, cols 4–7.
2 Orbach S. Commentary. *The Guardian* 1996; **May 31:** p 1, cols 4–6.
3 Garner DM, Garfinkel PE, Schwartz D, Thompson D. Cultural expectations of thinness in women. *Psychol Reports* 1980; **47:** 183–91.
4 Singh D. Adaptive significance of female physical attractiveness: role of waist to hip ratio. *J Pers Social Psychol* 1993; **65:** 293–307.
5 Bjorntorp P. Adipose tissue distribution and function. *Int J Obesity* 1991; **15:** 67–81.

Florid plaques and new variant Creutzfeldt-Jakob disease

SIR—Shutaro Takashima and co-workers (Sept 20, p 865)[1] describe a case of iatrogenic Creutzfeldt-Jakob disease (CJD) in a dura-mater-graft recipient, in whom the neuropathological features were characterised by the presence of prion protein (PrP) plaques surrounded by areas of spongiform change, the so-called florid plaque.[2] On the basis of this and two other similar cases, the investigators conclude that florid plaques are not specific to new-variant CJD (nvCJD), thereby raising questions about the neuropathological diagnostic criteria for this disorder.

Neuropathology is essential for the diagnosis of nvCJD, because the clinical features of this disorder are not specific and overlap with both sporadic CJD and other diseases during the course of the illness.[3] In our original report,[4] we did not claim that florid plaques were specific for nvCJD, since they were first described in a murine scrapie model,[2] and similar plaques are a prominent feature of the pathology of chronic wasting disease in mule deer and elk.[5] However, large fibrillary PrP amyloid plaques surrounded by a halo of spongiform change are characteristic for nvCJD and are easily recognised in routinely stained sections. The illustration by Takashima and co-workers shows PrP plaques stained by immunocytochemistry in an area of confluent spongiform change, which is not characteristic for nvCJD florid plaques in which the halo of peripheral spongiform change is typically discontinuous, probably representing dilated neuronal processes within otherwise intact neuropil.[2,4,5] Irrespective of the presence of florid plaques in their case, the other characteristic neuropathological features of nvCJD—spongiform change most pronounced in the basal ganglia, abundant PrP deposition in the occipital cortex and cerebellar molecular layer with perineuronal and perivascular deposits, and marked thalamic gliosis—were absent. These features allow the clear distinction of nvCJD from other varieties of human prion disease. Obviously, not all these features can be detected in small cerebral-cortical samples and diagnostic difficulties may be encountered in the interpretation of cortical biopsies from patients with suspected nvCJD. For this reason, we would not recommend brain biopsy as a reliable diagnostic investigation for nvCJD. Analysis of PrP glycoform provides a potential molecular marker for nvCJD, and frozen brain tissue should be retained from all suspected cases for this purpose. Although there is much current interest in diagnostic clinical studies[3] and tonsillar biopsy in the investigation of patients with suspected nvCJD, necropsy neuropathology is essential for diagnosis.

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1 Takashima S, Tateishi J, Taguchi Y, Inoue H. Creutzfeldt-Jakob disease with florid plaques after cadaveric dural graft in a Japanese woman. *Lancet* 1997; **350:** 865–66.
2 Fraser H. The pathogenesis and pathology of scrapie. In: Tyrell DAJ, ed. Aspects of slow and persistent virus infections. The Hague: Martinus Nijhoff, 1979: 30–58.
3 Zeidler M, Stewart GE, Barraclough CR, et al. New variant Creutzfeldt-Jakob disease: neurological features and diagnostic tests. *Lancet* 1997; **350:** 903–07.
4 Will RG, Ironside JW, Zeidler M, et al. A new variant Creutzfeldt-Jakob disease in the UK. *Lancet* 1996; **347:** 921–25.
5 Guiroy DC, Williams ES, Yanagihara R, Gajdusek DC. Topographic distribution of scrapie amyloid-immunoreactive plaques in chronic wasting disease in captive mule deer. *Acta Neuropathologica* 1991; **81:** 475–78.

Thrombolytic therapy for acute ischaemic stroke

SIR—As an observer of the stroke scene, I am struck by the circular argument in the recent review by Joanna Wardlaw and colleagues (Aug 30, p 607).[1] On the one hand, they argue that there is no objective evidence that tissue plasminogen factor (tPA) is safer than streptokinase, since the streptokinase trials were flawed by protocols modelled on the treatment of myocardial infarction in terms of adjunctive therapies, dosage, and timing. On the other hand, they present this flawed evidence as the basis for a systematic

review of thrombolysis in stroke, thereby diluting valid evidence in favour of tPA.

The NINDS[2] protocol was built on the hard-won experience of earlier trialists, and the results show unequivocally that tPA is effective and worthwhile for a subgroup of stroke patients, when delivered by expert teams. The question is how far beyond the strict NINDS protocol, which is difficult to implement in the wider clinical environment, can we go? As Wardlaw and co-workers acknowledge, this question is the object of continuing trials, including ECASS II; but this fact does not negate the proven value of tPA when given according to the NINDS protocol. As to streptokinase, I share the hope that well-designed trials will eventually show its efficacy, but those wishing to do so must assume the burden of proof. To square the circular argument, there is no objective evidence that streptokinase is as safe as tPA.

Finally, to argue that more than 50 000 patients were randomised before cardiologists became convinced of the benefits of thrombolysis in myocardial infarction is misleading. The benefit was clear after GISSI-1[3] for most patients with left-ventricular Q-wave infarction. The FTT[4] study was valuable in that it quantified the risk-to-benefit ratio in subgroups such as the elderly or patients treated more than 12 h after myocardial infarction. Similarly, NINDS has quantified the risk-to-benefit ratio for a subgroup of stroke patients. To dispute this now is unhelpful. The issue is how to refine the management of stroke patients such that appropriate patients can receive treatment.

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1 Wardlaw JM, Warlow CP, Counsell C. Systematic review of evidence on thrombolytic therapy for acute ischaemic stroke. *Lancet* 1997; **350:** 607–14.

2 The National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group: tissue plasminogen activator for acute ischemic stroke. *N Engl J Med* 1995; **333:** 1581–87.

3 GISSI Study. Effectiveness of intravenous thrombolytic treatment in acute myocardial infarction. *Lancet* 1986. i: 397–401.

4 Fibrinolytic Therapy Trialists' Collaboration. Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all randomised trials of more than 1000 patients. *Lancet* 1994; **343:** 311–22.

SIR—We have some objections to Wardlaw and colleagues'[1] conclusions. The observation that factors other than the agent used may account for the worst results attained with streptokinase as compared with tissue plasminogen factor (tPA) is an assumption, not evidence based, since no comparison of the baseline prognosis for patients enrolled in the two types of trials is reported. The only information we can gather from the article is that tPA is always better than streptokinase, not only for "composite outcome of death and dependency", which the investigators correctly regard as the most important, but also in terms of the early and late case fatality, whereas no significant differences in terms of intracranial haemorrhages are evident.

If 3435 patients are but a tiny fraction of the 4·4 million who die from a stroke each year, this is also true for 20 000 or 50 000 patients. Very large trials will never be able to enrol a study population that approximates the number of people who have a stroke every year, and such trials are necessarily less controlled and expose patients to unacceptable risks without reaching strong achievements. This happened, for example, for the International Stroke Trial,[2] which after randomising 20 000 patients reached the feeble conclusion of a small favourable effect of aspirin. On the other hand, 3435 patients may or may not be a relevant number depending on the quality of results—ie, how representative of the whole stroke population they are. At this time, we would still oppose large trials with thrombolysis, but a subsequent phase directed at evaluating effectiveness rather than efficacy should follow. However, we maintain that the correct philosophy in stroke therapy favours the splitters rather than the lumpers. In our view, this is the only way to effectively help our patients—a minority to begin with, but with a gradual extension supported by good science and health systems.

Finally, we question the need for such a review at this time. As Wardlaw and co-workers themselves remind us, new studies are running "and may; finish within a year". Hence, the conclusion that thrombolysis must be tested again not only adds poorly to many other similar statements, but is also too obvious since it is already practically applied by active investigators worldwide.

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1 Wardlaw JM, Warlow CP, Counsell C. Systematic review of evidence on thrombolytic therapy for acute ischaemic stroke. *Lancet* 1997; **350:** 607–14.

2 International Stroke Trial Collaborative Group. A randomised trial of aspirin, subcutaneous heparin, both or neither among 19 436 patients with acute, presumed ischaemic stroke. *Lancet* 1997; **349:** 1569–81.

SIR—The Cochrane Collaboration reviewers of thrombolysis in stroke[1] choose to draw conclusions that are not best supported by their data, and thus reinforce recent criticisms of meta-analysis.

In presenting their analysis of streptokinase and tissue plasminogen factor (tPA) trials, they seem to dismiss both pharmacological and statistical differences in order to conclude that there is no evidence that tPA is safer than streptokinase. More strangely, they exclude from the streptokinase overview half the MAST-Italy-trial[2] patients on the grounds of aspirin exposure, despite widespread aspirin use within the remaining trials, and in the absence of statistical evidence of heterogeneity. Indeed, a formal meta-analysis of the data on individual patients from the streptokinase trials available to the investigators fails to show a significant influence of aspirin (TAS-PP group, unpublished data).

The primary endpoint of the major recent trials was death and disability combined. On this endpoint, there is heterogeneity neither in the streptokinase trials (χ^2 2·33, degree freedom 4), nor among the tPA trials (2·38, 2), but the streptokinase trial results differ significantly from those of the tPA trials (15·08, 7, $p<0·05$). The 95% CI do not overlap, and reveal significant benefit with tPA but not streptokinase (tPA: odds ratio [OR] 0·57 [95% CI 0·45–0·72]; streptokinase: 0·98 [0·78–1·24]).

The proposal that further, larger clinical trials including streptokinase are required demands critical evaluation. Case fatality for all streptokinase trials, as presented in their figure 3, is clearly increased (OR 1·43 [1·10–1·88]), whereas that for tPA does not differ significantly from placebo (1·06 [0·80–1·39]). When the MAST-Italy figures are included, streptokinase comes out even less favourably (1·71 [1·35–2·16]), and the lower end of the CI barely touches that for tPA.

We agree that further trials and rigorous audit are required to determine whether the benefits of tPA can be translated safely into clinical practice. At present, the use of tPA even in clinical trials should be strongly discouraged. Despite previous enthusiasm in coordinating the Glasgow and MAST-Europe