incorporated into the model by use of a gamma function, with a mean that could change linearly over time. Approximate 95% CI were calculated by parametric percentile bootstrap from 499 samples.

By June 30, 2000, 75 cases of vCJD had been identified by the National CJD Surveillance Unit, 69 of whom had died. 59 cases had been confirmed neuropathologically. The other 16 cases, six of whom were alive, were classified as probable vCJD.

The annual numbers of clinical onsets, classifications, and deaths are shown in the table. Given that further cases with onsets in 1999 and 2000 will probably be identified in future months, the number of onsets clearly increases each year. Already, in the first 6 months of this year, 14 people have died compared with 18 deaths for the whole of 1998.

We estimated that the number of onsets increased by 23% per year for 1994–2000 (95% CI 7–42, p=0·004). The delay from onset to classification as probable or definite also shortened significantly (mean reduction 9% per year, p=0·014). The underlying incidence is estimated to have increased from two cases per quarter in 1994 to a current incidence of 6·5 cases per quarter (figure).

Dates of deaths were analysed by Poisson regression. Adjustment for reporting delays was unnecessary because most deaths are reported quickly. We estimate that deaths increased by 33% per year for 1995–2000 (8–64, p=0·005; figure). The high number of deaths in the fourth quarter of 1998 meant that the variation was greater than expected in a Poisson model. We adjusted for the extra variation when we calculated the CI and assessed the significance of the trend.

The incidence of a disorder can seem to increase if completeness of case ascertainment improves. Given the extensive publicity surrounding vCJD since its identification in 1996, however, we believe that this explanation for our findings is unlikely. The inclusion of living probable cases in our analysis will have contributed to our ability to detect the reported trend. There is, however, evidence of a significant temporal trend when the analysis is restricted to deaths. We believe that our findings reflect a real increase in the incidence of vCJD in the UK. Such an increase is clearly a matter of concern, although we emphasise that the absolute number of cases is low. We cannot tell how long the current increasing trend will continue and, therefore, have not predicted future numbers of cases.

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Endovascular treatment for dissection of the descending aorta

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Surgery for acute ischaemia complicating dissection of the descending aorta is associated with high mortality. We used an endovascular fenestration approach (scissor technique) to treat seven of 12 patients with ischaemic complications of descending aortic dissection; the remaining five patients were treated by stent implantation. Four of the 12 patients died (two in the fenestration group and two in the stenting group) in the days after the procedure. The remaining eight were symptom-free a mean of 9·4 (SD 8) months later. We suggest that the fenestration approach is a promising addition to endovascular treatment for patients with ischaemic complications of descending aortic dissection.

Dissection of the descending aorta is first managed conservatively because of the high mortality associated with surgical intervention.[1] However, in about 30% of patients, acute complications such as mesenteric, renal, or lower-extremity ischaemia, or chronic complications such as expansion of the false lumen, require intervention. In view of the poor prognosis after surgery, alternative endovascular techniques have been proposed.[2–4]

We report the endovascular treatment of 12

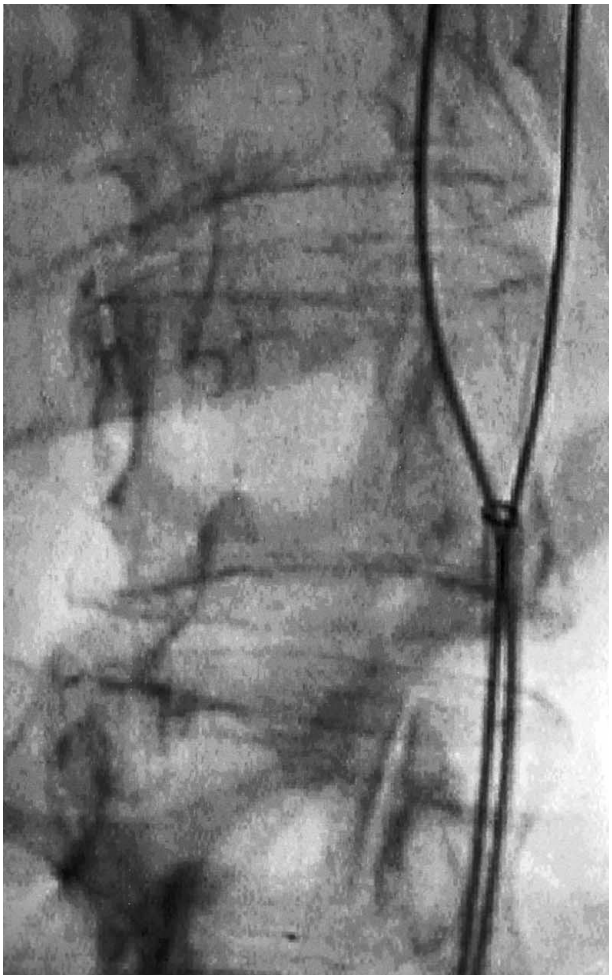

Figure 1: **Introduction of rigid guidewires into true and false lumens through same introducer sheath**

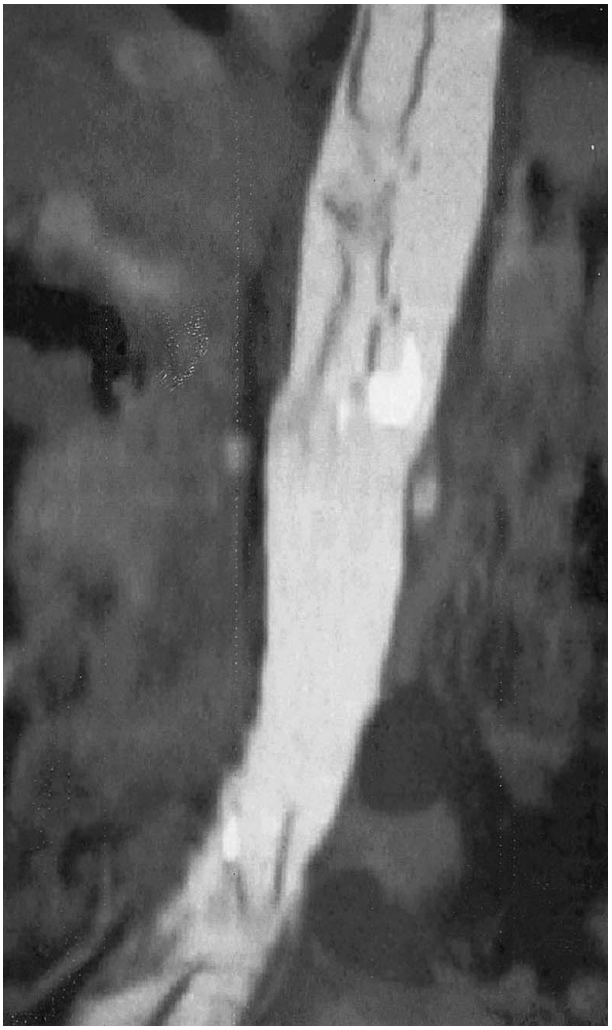

Figure 2: **Helical computed-tomography scan showing true lumen in centre of false lumen**
The intimal flap was torn from the distal abdominal aorta to the upper aorta; fenestration resulted in an endovascular endarterectomy but with the tissue left in situ. A long stent was placed in the left renal artery because of persistent static obstruction after fenestration.

patients (eight men) with a mean age of 53 (SD 14, range 22–68) years and ischaemic complications of descending aortic dissection (three with type A, nine with type B) confirmed by computed tomography (CT) scans or transoesophageal echodoppler ultrasonography. Six patients had signs of renal ischaemia; all had raised serum creatinine concentrations; two were anuric; six had acute lower-limb ischaemia; and seven had severe abdominal pain (with mesenteric ischaemia in six). For all these patients, our cardiovascular surgical teams felt that the risk of mortality associated with surgical intervention was prohibitive. The endovascular procedures were done under local anaesthesia and the first approach was via the femoral artery. An initial aortogram was done in a frontal projection with a 5 French catheter, which confirmed the findings of the echodoppler ultrasonography or CT scans concerning the degree of compression of the true lumen and the patency of the major branches of the aorta.

Static obstruction as a result of extension of the dissection into the lumen of an aortic branch vessel was encountered in five cases and was treated by stent implantation of the branch. When dynamic compression (compression of the true lumen by the false lumen resulting in ischaemia in multiple territories) was seen (seven cases), a new technique of fenestration that did not require perforation of the flap or balloon angioplasty was used. The technique was based on the insertion of two rigid guidewires, one in the true lumen and the other in the false lumen, through a single 8 F intro -ducer sheath (figure 1). The two guidewires were firmly anchored together and the sheath was inserted until it came into contact with the exit site of the dissection. The guidewires and sheath formed a pair of intravascular scissors that was then rapidly advanced over several cm to cut the flap. The angiogram done after the scissor manoeuvre showed one of three outcomes: a tear in the middle of the dissection flap (the ideal result [one patient]), a tear at the point where the dissection was attached to the aortic wall that left the dissection flap floating freely in the aorta (two patients); or the flap remained intact but the dissection extended to become circumferential, leaving the true lumen floating inside the false lumen (four patients). This latter mechanism was not associated with adverse consequences. All three mechanisms were equally effective in restoring flow to the hypoperfused branches, since they all permitted equalisation of the pressure in the two channels. Complementary stent implantation was done in three cases because of residual compromised flow (figure 2).

Four (two with stenting, two with fenestration) of the 12 patients died in the days after the procedure as a result of stroke (one), multisystem failure (two), or sudden unexplained death (one). In the remaining eight patients, the symptoms or signs that prompted the procedure all regressed in the days that followed; one of the two anuric patients required haemofiltration for 1 week, at which time his renal function had returned to normal. The eight patients who were discharged from hospital alive were symptom-free at a mean follow-up of 9·4 (SD 8) months. None had recurrent symptoms suggestive of hypoperfusion. Echodoppler ultrasonography or helical CT scans confirmed continued patency of all the treated arteries. Although the aortic diameter did not change over this short period, further follow-up will obviously be required.

Early endovascular intervention for acute ischaemic complications of descending aortic dissection, although still associated with substantial mortality, is a promising and evolving approach that may improve the poor prognosis in such patients. The techniques we describe are widely used and any interventional radiologist could carry out this procedure.

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