Court judge. He said: "Forget about drug deaths and acquisitive crime, about addiction and AIDS; all this pales into insignificance before the prospect facing the liberal societies of the West, like a rabbit in the headlights of an oncoming car. The income of the drug barons is an annual $254 thousand million, greater than the American defence budget. With this financial power they can suborn the institutions of the State and, if the State resists, with this fortune they can purchase the firepower to outgun it. We are threatened with a return to the Dark Ages of rule by the gang. If the West relishes the yoke of the Vikings, the Vandals or the school bully, current policies promote that end". The Attoney-General of Colombia, Sr de Greiff agreed with this statement, and called openly, publicly, and officially for the complete legalisation of all drugs (which is more liberal than my view[3]). Delegates from Italy concurred saying that it is "five minutes to midnight" and the world-wide gangsters must be deprived of this enormous financial muscle. A confidential report[4] to international law-enforcement agencies accords with this analysis. The Colombians ended with brave good humour saying that they had survived gunshot attacks by the cartels, adding "We were lucky. They seldom miss". Newcombe warned, even more ominously, "It's not five minutes to midnight, it's five minutes past". The behaviour of Clinton, Major, Mitterand, and other heads of state will show who is right.

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Bromocriptine and drug information

SIR—Bell (Oct 30, p 1118) refers to a perceived discrepancy between side-effect data presented for Parlodel (bromocriptine) in the UK *Data Sheet Compendium* and the US *Physicians Desk Reference*. It is important to point out, however, that both these publications are in fact in agreement in relation to the occurrence of headache in patients receiving bromocriptine therapy. Headache is, as Bell points out, referred to on the UK datasheet[1] as an occasional event in patients receiving bromocriptine for all indications. In a subsequent paragraph specifically relating to post-partum women treated with the product for the prevention of lactation, severe headache and transient visual disturbances are again referred to as possible precursors of seizure or stroke in this patients' subgroup. Every effort is made to ensure that the prescribing information provided to clinicians is consistent on an international basis.

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1 ABPI Data Sheet Compendium 1993–94. London: Datapharm, 1993.

Author's reply

SIR—I accept O'Sullivan's assertion that in the 1993/94 version of the *Data Sheet Compendium*, there is brief mention of the risk of serious problems in the use of bromocriptine (Parlodel) in post-partum lactating mothers. In 1994 we are still provided with less information than were American physicians in 1989 with respect to the use of Parlodel. Then, in America they had information, not only of the risks of strokes and seizures, but also of possible myocardial infarctions in association with the use of Parlodel: indeed this was emphasised in bold type. In the *Data Sheet Compendium* in 1990/91 there was no mention of this risk, whereas detailed information was being given in America in the *Physicians Desk Reference* of the previous year.

O'Sullivan makes my point, in that in England physicians are at risk of continuing to prescribe drugs when there is a potential association of severe risk, for years longer than their American counterparts, precisely because the information comes much delayed in the UK compared with America, and that when it does come the associated potential risks are still less emphasised and detailed.

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Length of published reports

SIR—On completion of a study most researchers want to get their results published at the highest possible level. Do most papers need to be as long as they typically are? Despite editorial attempts to limit article lengths, most authors make statements in the summary, repeat some of them in the introduction, describe them in the materials and results sections, and emphasise and elaborate on them in the discussion. Is all this repetition necessary? Do we as doctors or scientists need to have something repeated 2 or 3 times before we accept or understand it? We think not. We think that papers could be shortened without losing their message if much of the repetition was eliminated, details of standard methods were referenced, and the discussion confined to key elements of the study with a minimum of conjecture.

The Scots have a saying that "guid gear often comes in sma' bulk". Although articles in *The Lancet* are typically concise (mean 3·9, SD 1·1 columns in November, 1993, *vs* 4·2 [1·0] in the *British Medical Journal* [ns] and 5·7 [1·3] in the *New England Journal of Medicine* [$p < 0.001$]), one way that more articles, such as short reports, could be published each week would be to ask authors of full papers to reduce them by 300 or 600 words as a condition of publication; they can do this better than most. With more space available for short reports it would become easier to get one published.

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A novel marker for Borna disease virus infection

SIR—Borna disease virus (BDV) is a unique, negative, single-stranded RNA virus that preferentially infects limbic areas of the brain. Besides natural infections in horses and sheep, the virus can be transmitted to other mammals and even birds.[1] Clinical manifestations include (immune-mediated) encephalitis as well as non-fatal phasic behavioural abnormalities and almost symptom-free virus persistence.[1] Since antibodies have been found among patients with neuropsychiatric[2,3] and immunological disorders[3] in significantly higher concentrations than among healthy volunteers,[3] BDV is suggested to afflict people too. Moreover, a relation with limbic diseases in particular seems possible, as acute patients with major depression showed seroconversion and extremely high antibody prevalence (30%) at follow-up tests.[4] However, infection markers other than antibodies are needed as further proof.

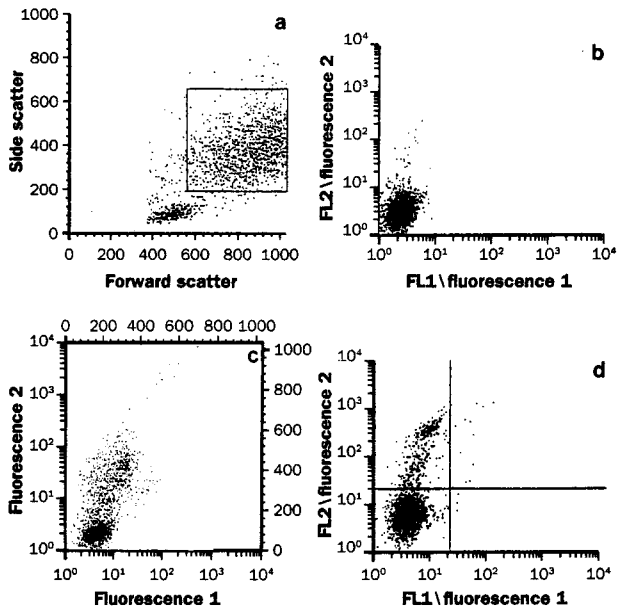

Figure: **Flow cytometric identification of human monocytes within PBMCs from whole blood (a) and detection of BDV antigens in monocytes from acute (c) and chronic (d) psychiatric patients**

(a) Scattergram of PBMCs shows gated monocytes compared with non-gated cells below (lymphocytes) corresponding to their size (forward scatter) and granularity (side scatter). (c-d) Dot-plots of patients' monocytes after labelling with BDV-specific monoclonal antibodies; (b) with control monoclonal antibodies, and all with PE (orange fluorescence 2–FL2) anti-mouse IgG (Sigma). Antigen-positive subpopulations (15–17%) are shown in (c), antibody-negative 56-year-old woman 3 weeks after onset of acute episode of major unipolar depression; and in (d), antibody-negative 41-year-old man with 19-year history of organic mood disorder and epileptic seizures due to cerebral haemorrhage, during depressive episode. In (d), negative and positive cells are divided by quadrant statistics. Fluorescence 1(FL1) represents green fluorescence, here shown as autofluorescence.

We present the first data to our knowledge on the detection of BDV antigen in peripheral blood monocytes (PBMs) from psychiatric inpatients, by use of flow cytometry[5] done with a FACScan (Becton-Dickinson, San Jose, California). Peripheral blood mononuclear cells (PBMCs) were isolated from citrate-treated blood by centrifugation on Ficoll-paque (density 1.077), fixed with paraformaldehyde, and treated with Triton X-100. PBMs were identified within PBMC populations by a scattergram analysis according to size and granularity (figure, a) and controlled by CD14 fluorescence. Contaminations of the gated monocytes with either CD67 neutrophilic granulocytes or CD4/CD8/CD19 lymphocytes were excluded. BDV antigens were investigated with monoclonal antibodies against the major viral proteins (38/40 kDa and 24 kDa)[1] and phycoerythrin (PE) labelled anti-mouse IgG.

From cohorts with either acute[4] or chronic psychiatric patients, dot-plots of typical antigen carriers showed a subpopulation (15–17%) of monocytes, not lymphocytes (data not shown), that was clearly stained by BDV-specific monoclonal antibodies (figure, c and d), but not by control monoclonal antibodies (figure, b). Among each cohort (70 patients), we detected a mean rate of antigen carriers of 40–50% by FACS analysis, which was considerably higher than the mean prevalence of antibody carriers (20%) by immunofluorescence.[4] Positive monocytes were also found in naturally or experimentally infected animals. We conclude that viral antigen in PBMs commonly indicates BDV infection. It can thus be regarded as a novel in-vivo marker that allows reliable diagnoses independent of the host's actual antibody responses.

This marker confirms the ability of BDV to infect people. Its high frequency in certain mental disorders may indicate a relation with disease. Furthermore, peripheral antigen-positive cells represent a unique tool in search of the human BDV genome.

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Creutzfeldt-Jakob disease and blood transfusion

SIR—Esmonde and colleagues[1] described 29 patients with Creutzfeldt-Jakob disease (CJD) who donated blood before manifestation of the disease. They excluded a possible risk of transmission of the disease from geographical data that showed no increase in incidence of CJD in areas where these donors lived at the time of donation. However, potential problems with their approach are that it is unclear when viraemia occurs during the disease[2] and the incubation period, as observed in iatrogenic CJD from human growth hormone therapy (mean 5–17 years), is highly variable.[3] Because recipients of blood transfusions are usually critically ill and, therefore, have a disturbance of the blood-brain barrier, we hypothesise that CJD may reach the nervous system during the viraemic phase of CJD. The possibility of transmission by blood transfusion can only be ruled out by follow-up of each blood unit donated from a patient with CJD.

We identified a 62-year-old man who died from CJD in 1991. Between 1971 and 1991, he had donated 55 blood units. We traced the records of these units and identified 27 patients who definitely and 8 who probably received a blood unit from this patient. The recipients of 20 units could not be determined. 18 of the patients who received a blood transfusion had died. All died from medical reasons (tumour, transplant failure, myocardial infarction, or pneumonia). No one died from dementia or a neurological cause and no one had had signs of dementia or mental deterioration. The mean survival time was 1·4 years (range 0–14 years). 9 patients were still alive; their survival times were 4, 5, 8, 11, 12, 12, 16, 17, and 20 years. The patients and their general practitioners were contacted by telephone. 1 woman had had a stroke 10 years ago. All others