severe abdominal pain. 3 patients treated with glucose subsequently underwent hysterosalpingography showing normal configuration and patency of the affected tubes.

We feel the results of this study are promising and that treatment with glucose could be a useful alternative to conventional methods.

Steroid-Induced Psychosis

Sir,—Dr d’Orban (Sept 16, p 694) rightly draws attention to the unsatisfactory state of the law with respect to steroid-induced psychosis as a defence for criminal activity, but is not quite accurate in stating that there is only one other publication on this matter. A 15-year-old man has been reported, who, following treatment with prednisolone 30-60 mg daily for severe ulcerative colitis, was convicted of shop lifting despite clear evidence of a steroid-induced psychosis.

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MEAN LEVELS OF PLASMA FIBRINOGEN, PLASMA VISCOSITY, AND WHITE CELL COUNT (WCC) IN MEN AGED 45-59 YEARS

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participants gave informed consent—and from blood donors at the National Institutes of Health blood bank whose blood was designated for research purposes. The four groups (table) were: 

**Group 1.** Healthy Jamaican HTLV-I seronegative, n = 15. Randomly chosen from a nationwide survey of HTLV-I antibody in 1985-86. Enrolled when seeking employment licences for food-handling. Questionnaire and physical examination were administered by trained personnel.

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**Group 4.** Healthy USA control, n = 13. Healthy blood donors all seronegative for HTLV-I (and HIV-1).

Sera were tested for antibody to HTLV-I with a research ELISA based on disrupted whole virus particles (Dupont). Positive samples were confirmed by Western blot (Biotech). Minimum criteria for a positive Western blot was bands specific to HTLV-I gag proteins p19 and p24. An in ELISA (Cambridge Bioscience) was also done—i.e., antibodies to two HTLV-I gene groups were required for a positive sample.

Cryopreserved lymphocytes were plated in triplicate at a concentration of $3 \times 10^5$ per well in 96-well plates. We used RPMI medium supplemented in 2% human AB serum. After 4, 5, and 6 days, wells were pulsed with 1 μCi of $^3$H-thymidine for 4 h, harvested (Skatron, Sterling, Virginia), and counted. Spontaneous lymphoproliferation counts were log-transformed and analysed by logistic regression. Positive samples were confirmed by Western blot (Biotech). Minimum criteria for a positive sample were: evidence of difference by age and risk group. Optimal r determinants included variables influenced by exposure to antigens and pathogens; and multiple infections were more common in Jamaica than in the United States. Participants gave informed consent—and from blood donors at the National Institutes of Health blood bank whose blood was designated for research purposes. The four groups (table) were: 

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Spontaneous lymphocyte proliferation rates at day 6 are summarised in the table. The important significant differences were between seropositive and seronegative healthy Jamaicans, seropositive healthy Jamaicans and seropositive patients with ATL, and seronegative Jamaicans and US controls. There were no correlations between proliferation rate and white blood cell or lymphocyte counts.

Among the 30 healthy Jamaicans, the only demographic feature (age, sex, socioeconomic status) associated with high lymphoproliferation was low income. However, logistic regression analysis revealed that HTLV-I seropositivity was a stronger risk factor for high lymphoproliferation (OR = 17.2, 95% CI 8-18-6) than was low income (OR = 8-5, 95% CI 0-9-81-6).

Besides ATL and HAM/TSP, HTLV-I infection has been associated with immunodeficiency and in-vitro immunological effects,14 including decreased lymphocyte proliferation in cell cultures.15 Our data suggest that increased spontaneous lymphoproliferation is associated with HTLV-I exposure per se. Since the rate of proliferation was indistinguishable in HTLV-I positives and controls, the low spontaneous lymphocyte proliferation in ATL probably reflects an overabundance of HTLV-I-positive tumour cells that have lost their capacity to proliferate. Individuals co-infected with HTLV-I and HIV-1 may progress to AIDS at an accelerated rate.40 HTLV-I lymphocyte proliferation may explain this. HTLV-I-negative black Jamaicans had significantly higher lymphoproliferation than US white controls. The reason for this finding is unclear. Genetic factors may account for these differences.41 However, it is also possible that spontaneous proliferation is influenced by exposure to antigens and pathogens; and multiple infections are more common in Jamaica than in the United States.