the clinical manifestations". The morphological lesions in the case described here point to the reverse. Our findings suggest that substantial functional reserves exist within the striatal system where considerable morphological damage does not find expression in clinical symptoms. One feels tempted to draw a parallel with Parkinson's disease where a loss of 80–85% of nigrostriatal neurones is necessary to precipitate clinical symptoms.[3] A morphometric analysis on the brain will be published later.

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UNUSUAL SEROLOGICAL PROFILES IN AIDS

SIR,—Dr Dalgleish and colleagues (April 19, p 911) report a patient whose antibody profile by radioimmunoprecipitation (RIP) is interpreted as possibly indicating infection with a "variant" virus serologically distinct from the HTLV-III/LAV prototype. A child with cryptococcal meningitis, severe wasting, and thrombocytopenia had been given a blood transfusion years earlier from a donor who had visited North Africa. The patient's sera reacted by RIP to HTLV-III/LAV envelope proteins only. However, such reactivity is common in patients with AIDS. As many as 50% of AIDS patients lack detectable antibodies to the *gag*-related proteins p24, p55, and p17 in RIP, while maintaining antibodies to *env*-related antigens.[1] In addition, *gag* gene proteins of related viruses are more conserved and thus more immunologically cross-reactive than are exterior glycoproteins, which contain many type-specific antigenic determinants. For example, as has been observed for a recently reported human retrovirus from Africa (HTLV-IV), cross-reactivity on RIP indicative of a distinctly different viral infection is seen primarily to *gag* products p24 and p17 and to *pol* products p64, p53, and p34 rather than to the envelope proteins gp160 and gp120.[2] It is more likely, therefore, that the patient presented had infection with the HTLV-III/LAV prototype rather than a new or related retrovirus.

The ELISA reactivity reported for this patient was consistently negative. Although this could be a result of infection with a virus other than HTLV-III/LAV, it is more probable that it represents a drawback of the ELISA test in certain clinical situations. This patient was receiving intermittent high-dose glucocorticoids.[3] Patients taking immunosuppressive drugs (because of cancer or an organ transplant) may be ELISA negative for weeks or months while reacting positively for HTLV-III/LAV by other, more sensitive confirmatory tests (R. M., unpublished).

We suggest caution in interpreting less common serological profiles as indicating infection with a new or as yet unclassified virus when the results are more easily explained as being consistent with infection with HTLV-III/LAV.

Although HTLV-III/LAV variants do exist in Africa the term "variant" can be confusing. Many isolates of HTLV-III/LAV have been identified and analysed by molecular cloning techniques and, though considered variants by restriction enzyme analysis, they are serologically indistinct from HTLV-III/LAV. Isolates of HTLV-IV do seem to be serologically indistinct from the Simian T-lymphotropic virus type III of African green monkeys ($STLV\text{-}III_{AGM}$). Yet, as previously mentioned, immunological cross-reactivity to HTLV-III/LAV exists, especially to the *gag*-related antigens. This "cross-reactive variant" is a distinct virus and can be evaluated on the basis of the degree of serological cross-reactivity with an HTLV-III/LAV antigen source or with an STLV-III or HTLV-IV antigen source. This divides the African isolates of human T-lymphotropic viruses into at least two groups. If individuals with unusual antibody reactivities were to be screened by immunoblotting or by immunoprecipitation with the above viruses the profiles would fall into one of two categories: those whose antibodies react strongly to the HTLV-III/LAV envelope proteins (gp160, gp120, and gp41) and those whose antibodies react to a higher degree with the envelope proteins of STLV-III or HTLV-IV (gp160/120 and gp32). This protocol would help clarify whether a particular seropositive patient was in fact infected with an HTLV-III/LAV virus or with a related, cross-reactive virus. Since HTLV-IV does not seem to be associated with AIDS, identification of infecting virus may then have future clinical relevance.

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HEAT TREATMENT OF FACTOR VIII CONCENTRATE

SIR,—White et al[1] describe seroconversion to antibody positivity to human immunodeficiency virus (HIV) after the use of factor VIII concentrate heated in a lyophilised state, and van den Berg and colleagues[2] report another such incident. McDougal et al[3] have demonstrated that inactivation of HIV is a function of the matrix in which the virus is contained, the temperature employed, and the duration of time for which that temperature is applied. Inactivation in a liquid matrix is more efficient and swift than that achieved in a lyophilised state. Similarly, heating at a lower temperature or for a shorter duration is less efficient than heating at a higher temperature for longer. Reports indicating transmission of active HIV should therefore indicate in reasonable detail the duration of heat treatment, the temperature applied, and whether the preparation was in a liquid or lyophilised state during such treatment, to allow meaningful conclusions about the safety of heated preparations. After all, heat treatment could be said to have been accomplished if a product is heated to, say, 40°C for 30 min.

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★★Dr Gilbert C. White II (Durham, North Carolina) and Dr W. van den Berg (Amsterdam) have informed us that heat treatment was, in both cases, at 60°C for 30 h in a lyophilised state.—ED. L.