From these three reports, we conclude: 1 Intravenous immune globulin can induce aseptic meningitis in children as well as in adults: the strong temporal association (on day three in our patient and in Watson's patients) and the repetitive occurrence in the same patient (on two occasions in Kato's patient and in our patient) are both strong arguments in favour of this. It should no longer be a hypothesis. 2 Several preparations of intravenous immune globulin can induce aseptic meningitis. Up till now it has been reported with four different preparations: with immunoglobulin prepared with polyethylene glycol and with a sulphonated preparation, with a formulation prepared by cold ethanol fractionation (Sandoglobulin) and with a low PH formulation prepared with ethylene glycol (Intragam). 3 As aseptic meningitis occurs in at least two other intratigogenic diseases, the aseptic meningitis in itself is rather benign and resolves rapidly with cessation of the therapy. But not recognising this complication as such might result in potentially dangerous and/or unnecessary examinations (lumbar puncture in a thrombocytopenic patient; CT scan).

The possible occurrence of aseptic meningitis following immune globulin infusion should thus be known by all physicians and should be mentioned by the pharmaceutical firms.

**The brain in schizophrenia**

The excellent editorial by Ron and Harvey notes that "to have forgotten that schizophrenia is a brain disease will go down as one of the great aberrations of twentieth century medicine". However, it is true to question as to whether schizophrenia can be considered as a brain disease in the same way as established brain diseases such as viral or atrophic disorders of the CNS. There may be more general reservations with the validity of the concept of schizophrenia itself, but I have four specific reservations with calling schizophrenia a brain disease:

1. Unlike many models, there is as yet no diagnostic pre- or post-mortem biological or other physical marker for schizophrenia.
2. Compared to most brain diseases, there is no predictable pattern of deficit in sensory or motor functions or in "primitive" reflexes.
3. Unlike most brain diseases, psychological or psychosocial variables play a significant part in the aetiology and suitability of outcome of many patients who have schizophrenia.
4. The relationship between neurological features of patients with schizophrenia and the pattern or severity of psychiatric disturbance is much more equivocal than in the case of analogous relationships in brain diseases.

At present I would therefore feel comfortable in calling schizophrenia a brain dysfunction, but I do not think there is yet sufficient evidence to call it a brain disease. It is possible that the term disease, if commonly applied to schizophrenia, may in the perception of some clinicians limit the range of viable therapeutic options.

**References**


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**References**


**Conclusions**


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