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 never 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, 1 with a formulation prepared by cold ethanol fractionation (Sandoglobulin) 2 and with a low PH formulation prepared with ethylene glycol (Intragam). 3 As with many other iatrogenic diseases, the pathogenetic mechanism of aseptic meningitis is not known. Arguments for and against the possible causality of aseptic meningitis have been put in the data sheets. A treatment form was also filled out, and this included questions about any unusual event during treatment.

Five time-related events occurred during these 370 infusions; blood pressure dropped but did not need any treatment (2), dyspnoea treated with a diuretic (1), temperature increase treated with clemastine (1) and transient macroscopic haematuria (1). The treatment course was not interrupted in any of the cases. The last event may have been coincidental, but the others may have been caused by the IgIV infusions. They were, however, mild and transient. Aseptic meningitis was not observed in any of the patients. We conclude that the incidence of side effects of high dose IgIV is low and that IgIV may be safely applied in neurological patients.

The brain in schizophrenia

The excellent editorial by Ron and Harvey 5 notes that "to have forgotten that schizophrenia is a brain disease will go down as one of the great aberrations of twentieth century medicine." 6 However, it is open 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 most other medical illnesses, there is 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 psychiatric variables play a significant part in the aetiology and stability of outcome of many patients with schizophrenia.

4) The relationship between neurobiological 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.

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(”To”)-HM-PAO SPECT and dementia in Parkinson’s disease

With great interest we read the article from Spampinato et al 7 and we would like to comment on some aspects of the article. We performed single photon emission computed tomography (SPECT) using (”To”)-HM-PAO as a tracer in five clinically demented and 6 non-demented patients with Parkinson’s disease (PD). HM-PAO uptake was measured in the frontal, parietal and temporo-parietal cortex and was expressed as cortical/cerebellar ratio. The HM-PAO uptake in our patients had a reduction of HM-PAO uptake of more than 20%, which seems to be in concordance with Spampinato et al. In contrast Spampinato et al, however, found no difference between the demented and non-demented PD patients. We investigated whether there was a relation between SPECT-scan data and neuropsychological tests in our group as a whole (n = 11). Using the Spearman rank correlation test, we found no significant correlations between neuropsychological performance and SPECT data on any test.

One of the main problems in our research was the estimation of dementia in PD. Of the 5 patients who fulfilled the criteria for dementia as described in the DSM-III-R, all lacked any typical cortical features. All of our patients were able to undergo the neuropsychological tests. In the study by Spampinato et al, diagnosis of dementia was based on neuropsychological assessment, according to which the PD patients were divided into two groups of 15 patients. However, five of their patients could not be tested, probably due to severe dementia. It was not stated by the investigators whether their demented PD patients were free from cortical features.

In our opinion, one of the possible explanations for the differences in results is that patients had no cortical dysfunction and therefore no Alzheimer-like pathology. Further elucidation of dementia as seen in PD seems mandatory.

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The brain in schizophrenia.

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