MANN was she with comments.

Swelling parotid swelling may production spasm. 5

Flow and the patient clearly confined diffusion and "chemodenervation" of the glands producing watering tympanic eyes is not known to toxin injection similarly to that of botulinum toxin in parotid glands involving the unexplained. 1

Increased saliva injections for this patient’s symptoms of botulinum toxin in parotid glands was partly responsible for the production of saliva. 2

Mann’s patient clearly had a dry mouth as a result of the toxin action. Duct paralysis best explains the combination of symptoms and signs in this patient. 3

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Immunoglobulin treatment in human and experimental epilepsy

The paper of van Engel et al. mentions some positive effects of intravenous immunoglobulins (IVIg) in the treatment of refractory epilepsy. No reference about our experience in that field is mentioned, however, in the main literature reviewed by Van Engel et al. 4

In 1983, we successfully treated a patient with Lennox-Gastaut syndrome with IVIg for four months but relapsed afterwards although their seizures were less severe than before the infusions. In this open study, 15 patients with Lennox-Gastaut syndrome and partial epilepsy were infused with IVIg. 5 This treatment gave excellent results in two patients, who were seizure-free for months but relapsed afterwards although their seizures were less severe than before the infusions. In this open study, 15 patients with Lennox-Gastaut syndrome and partial epilepsy were infused with IVIg. 6 It was concluded that IVIg treatment may be very helpful not only in West and Lennox-Gastaut syndromes, but also in partial epilepsy, including Rasmussen’s syndrome. 7

At that time, however, all studies published about IVIg in refractory epilepsy were open designs — with the exception of that of Illm et al. 8 which was a single blind, cross over trial — with controversial schedules and doses. Indeed the patients received from two to more than 10 infusions with doses ranging from 100 mg to 1 g/kg/perfusion and no relation was assessed between dose or schedule of IVIg and clinical responsiveness. An overview of the medical literature involving about 200 epileptic patients treated with IVIg showed a positive response to this treatment in around 30% of the patients. 9 Taking that into account, in 1989 we initiated the first double blind study to establish a dose of IVIg for treatment of epilepsy. 10 Sixty one patients were randomly assigned to receive either IVIg (n = 43) or a placebo (n = 18) at three different doses (100, 250, or 400 mg/kg/infusion). No dose effect was found (P = 0.31). The data for the whole study population showed an improvement in 52-5% of patients treated with IVIg in accordance with previously reported open studies, compared with 27-8% in the placebo group; this positive trend was not significant (P = 0.09). When only the patients with partial epilepsy were assessed, a significant difference in favour of the IVIg treatment was found (P = 0.04) and this was confirmed in the subgroup of partial epilepsy with secondarily generalised seizures (n = 30) regardless of the dose (P = 0.04). Two patients became seizure free. One with Lennox-Gastaut syndrome needed also further anti-convulsant medication. The other, who had partial epilepsy, relapsed but is still better than before the IVIg.

The mechanisms of action are unknown. We found some relation between a lower serum IgA level and a better clinical response in the first study, 4 but could not confirm this correlation in the double blind study although we noted a trend in favour of a lower serum IgA level. 5 IVIg in refractory epilepsy are well tolerated but the major problems related to this treatment concern its cost and the hazards of transmission of infectious diseases or antibodies to blood derivatives. Immunoglobulins may be considered safe however, as their manufacturing procedures are known to inactivate human pathogenic viruses such as hepatitis A, B and C, and HIV. 11

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Van Engel et al reply: We thank van Rieckevorsel and Delire for their interest in our paper on immunoglobulin treatment in human and experimental epilepsy. Their point was that we did not mention their experience in that field. Our paper was an overview of some aspects of immunoglobulin effects in human and experimental epilepsies; it was not a review of the medical literature on immunoglobulin treatment in human epilepsies. We wrote a 1993 review on current immunoglobulin treatment in human epilepsies, and there we recognised their contribution in the field by citing three papers published by van Rieckevorsel and colleagues.

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