Localised autonomic failure due to botulinum toxin injection

I read with great interest Mann's report of a patient receiving botulinum toxin injections for spasmodic torticollis who developed sialoactasis and swelling of the parotid glands after each set of injections.1 Local diffusion of the toxin and paralysis of the smooth muscle of the parotid salivary ducts was proposed as a possible underlying mechanism.1

The inhibitory action of botulinum toxin is not confined to the neuromuscular junction. All the autonomic cholinergic fibres including the major secretomotor fibres to salivary glands are similarly blocked. Local diffusion and "chemodenervation" of the parotid glands leading to reduction of salivary flow and the development of chronic recurrent peribuccal sialorrhoea is a more likely explanation for this patient's symptoms. Dickson and Shevky in 1923 showed that tympanic nerve-induced salivary flow was variably flow and the development of chronic recurrent parotitis seems to be a more likely explanation for this patient's symptoms. 3 Dickson and Shevky in 1923 showed that tympanic nerve-induced salivary flow was blocked by the toxin in cats.1 In botulism, dry mouth is a common symptom, occurring in about 90% of patients. Dry mouth has also been reported in some 30% of patients after cervical injections for spasmodic torticollis.1 Paradoxically, excessive salivation has long been known to occur in patients with essential blepharospasm and hemifacial spasms.2 This paradoxical effect of the toxin on the "neuroglandular junction" remains unexplained. Increased salivation may partly be responsible for parotid swelling after botulinum toxin injections in the patient reported.

Immunoglobulin treatment in human and experimental epilepsy

The paper of van Engelshoven et al2 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 medical literature reviewed by van Engelshoven et al.2

In 1983, we successfully treated with IV Ig a patient with severe Lennox-Gastaut syndrome who still remains seizure free.3 Thereafter, in a first open study, 20 patients with Lennox-Gastaut syndrome and partial epilepsy were infused in order to treat IV Ig.4 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 have partially improved including eight with a pronounced decrease of seizures. It was concluded that IV Ig treatment may be very helpful not only in West and Lennox-Gastaut syndromes, but also in partial epilepsy, including Rasmussen's syndrome.5

At that time, however, all studies published about IV Ig in refractory epilepsy were open designs—with the exception of that of Illi et al,6 which was a single blind, cross-over trial—with constant IV Ig 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 IV Ig and clinical responsiveness. An overview of the medical literature involving about 200 epileptic patients treated with IV Ig showed a positive response to this treatment in around 50% of the patients.7 Taking that into account, in 1989 we initiated the first double blind/dose finding clinical study.8

We thank van Rijckevorsel and Delire for their interest in our paper on immunoglobulin treatment in human and experimental epilepsy. They were right that we did not mention their experience in that field. Our paper was an overview on 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 and experimental epilepsies, in which we recognised their contribution in the field by citing three papers published by van Rijckevorsel and colleagues.9

8 Van Engelshoven et al reply: 1

Matters arising

from swabs and absent response to broad spectrum antibiotics.

The concept of paradoxical hypersecretion is intriguing but one would not expect a dry mouth to result. Duct paralysis best explains the combination of symptoms and signs in this patient.

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van Engelshoven et al reply: 2

We thank van Rijckevorsel and Delire for their interest in our paper on immunoglobulin treatment in human and experimental epilepsy.1 Their point was that we did not mention their experience in that field. Our paper was an overview on 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 and experimental epilepsies, in which we recognised their contribution in the field by citing three papers published by van Rijckevorsel and colleagues.9