W135 and Y) meningococcal vaccine. Meningococcal strains can be separated into twelve serogroups, based on their capsular structure. Serogroup B is the most frequent isolated strain. Platonov has vaccinated 18 LCCD patients of whom 2 experienced a meningococi disease, 9 and 12 months after vaccination respectively. Unfortunately, the meningococcal strains of the 2 patients were not serogrouped and characterized further. Because it is very likely that the meningococcal involved had another serogroup than one of the four serogroups included in the vaccine (A, C, W135 or Y), we can not support the conclusion that these cases represent vaccine failures. In the same study, Platonov found a lower frequency of meningococcal disease in a period of 3 years after vaccination than before the vaccination, suggesting a beneficial effect. We have immunized 21 LCCD individuals with the tetravalent meningococcal vaccine and found that 2 patients developed meningococcal disease after vaccination [1]. One C8β-deficient 19-year-old female patient twice developed meningococcal disease with serogroup B (not included in the vaccine), 1 and 3 years after vaccination respectively. The first episode was due to meningococcus B:4:P1.4, and in the second episode a meningococcus B:4:P1.6 was isolated. The second 21-year-old C8β-deficient male patient developed meningococcal disease with serogroup Y (included in the vaccine), but the onset of the disease was more than 3.5 years after vaccination. This patient had developed a significant antibody response to serogroup Y (measured by ELISA) determined 6 months after vaccination. Meningococcal capsular polysaccharides do not activate T-helper cells and induce relatively poor memory. After vaccination, a protective period of 4 years is assumed. Therefore, LCCD patients at risk for meningococcal disease require revaccination each 3–3.5 years with the tetravalent vaccine. The use of vaccine that also confers immunity to serogroup B should be investigated further.

We agree entirely with Cremer and Wahn that LCCD patients should be informed about their risk to contract meningococcal disease in order to lower the threshold for early antibiotic treatment. However, for screening of complement deficiencies we strongly recommend to use also the more sensitive haemolysis-in-gel-assay and not the traditional CH50 and AP50 test only [2].

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Severe myoclonic epilepsy in infancy and carbamazepine

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Abbreviations *CBZ* carbamazepine · *SME* severe myoclonic epilepsy in infancy

Sir: Severe myoclonic epilepsy in infancy (SME) is a severe epileptic disorder proposed as a separate entity by Dravet et al. in 1978 [2] which has been regarded as one of the most intractable epilepsies in infancy and childhood. Because this disorder features both generalized and focal seizures [1], it has seemed reasonable to use the antiepileptic drugs which are appropriate for partial seizures including carbamazepine (CBZ).

Within the last 5 years, we have treated seven children with SME. Mean onset of the illness was around 5 months of age. Perinatal events were unremarkable. Precipitating factors of the seizures were mild fever, taking a bath and watching television or other flashing light sources. All patients had frequent episodes of prolonged convulsive seizures more than 30 min and also exhibited partial seizures in addition to other types of seizure. The seizures of these patients were extremely difficult to control. In six of seven patients, we administered CBZ. It was not effective. On the contrary, it aggravated generalized seizures in at least four of the six patients. Since it is difficult to make a correct diagnosis of SME early in the phase of illness, the attending physician may prescribe CBZ for partial seizures. If these seizures are aggravated by CBZ, SME may be suspected. Once a diagnosis of SME has been established, CBZ should not be administered. Our experience is in accord with that of others [3].

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