prednisone for 10 weeks. After this treatment the patient's condition improved slowly but continuously. At the time of the last clinical control examination 18 months after hospital discharge the patient was able to work without physical impairment.

Clinical and electrophysiological findings met all the criteria for the diagnosis of motor neuron disease. Clinical signs of lower motor neuron involvement were present in both arms. Electromyographic studies disclosed axonal loss at three different levels—namely, lumbar (anterior tibial muscle), cervical (hand muscles), and supraspinal (masseter muscle). Clear signs of damage to the upper motor neuron were also present. Although the symptoms of the patient could be explained by cervical myelitis the EMG findings with evidence of axonal damage in the anterior tibial and masseter muscle as well as the lack of any sensory abnormalities argue strongly against this possibility.

In addition, signs of inflammation in the CSF were not consistent with a diagnosis of amyotrophic lateral sclerosis. We identified a *Borrelia burgdorferi* infection of the CNS as the cause of the inflammation. Evidence included a raised specific IgG and IgA antibody index, the demonstration of *Borrelia burgdorferi* specific oligoclonal IgG bands in the CSF and the predominance of individual *Borrelia burgdorferi* specific antibody bands in CSF (as indicated by western blotting). The absence of a high white cell count and protein in the CSF could be attributed to prior antibiotic treatment. Optimising dose and duration, antibiotic treatment was renewed and combined with a long term steroid therapy. Four months later a CSF examination showed a considerable decrease in specific antibody concentrations, and the patient's condition continued to improve.

In the light of the evidence, it seems safe to conclude that the patient's symptoms were due to a CNS *Borrelia burgdorferi* infection which merely mimicked amyotrophic lateral sclerosis. Several reports have been published on spirochetal diseases leading to isolated damage to the motor system. Spinal meningovascular lues has been reported to cause a clinical syndrome mimicking motor neuron disease.[4] Fredrikson and Link published a case report of a patient with isolated upper motor neuron symptoms due to CNS borreliosis who responded favourably to antibiotic treatment.[5] Cases of painful motor neuropathy due to *Borrelia burgdorferi* specific infection have also been reported.[1] Halperin *et al*[6] found serological evidence of exposure to *Borrelia burgdorferi* in nine of 19 patients with motor neuron disease. However, none of them showed signs of *Borrelia burgdorferi* specific immunoreactivity in the CSF or favourable response to treatment.

It can be speculated that the spirochete *Borrelia burgdorferi* has the ability to induce an immune reaction that specifically affects motor neurons. This reaction may mimic different, non-curable diseases, such as spastic spinal paralysis, spinal muscle atrophy, and amyotrophic lateral sclerosis. Therefore, we suggest that patients diagnosed as having progressive motor neuron disease, who live in endemic areas, should be tested for *Borrelia burgdorferi* specific antibodies in serum and in CSF. The test could reliably detect a rare, but treatable disease mimicking motor neuron disease.

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Severe but transient parkinsonism after tetanus vaccination

A 38 year old metal worker with a history of hypertension and hyperthyroidism presented with fluctuating fever and sweating, palpitations, tremor of the upper parts of both legs, and diplopia. These symptoms had been present for five days and had started within hours after he had received the last of three vaccinations for tetanus (TE anatoxal berna, contents: 20 LF tetanus toxoid, 2 mg aluminium phosphate, and 0.1 mg thiomersal, Primmed BV, Almere, The Netherlands). These vaccinations were given because of an injury to his right index finger one month before. There was no family history of movement disorders. Physical examination showed profuse sweating, normal consciousness, a temperature of 37.3°C, symmetric rigidity of all four limbs, and a painful tremor in the upper parts of his legs. Muscle strength, tendon reflexes, and sensation were normal.

Within a week he progressed to severe hypokinetic dysarthria, a mask-like face, and a resting tremor of both hands, and he had bradykinesia and generalised rigidity, together with a cogwheel phenomenon in the arms.

Laboratory examination showed a creatine phosphokinase activity of 2682 U/l (normal <190 U/l) and normal blood concentrations of manganese, copper, ceruloplasmin, and carbon monoxide. His CSF showed 50 lymphocytes (normal range 0-3), slightly raised total protein (0.54 g/l; normal range 0.15-0.45 g/l), normal IgG index (0.43; normal <0.66), and negative serological tests on Epstein-Barr virus, herpes zoster virus, herpes simplex virus, syphilis, *Borrelia burgdorferi*, and *Mycoplasma pneumoniae*. Brain MRI was normal. Single photon emission computed tomography (SPECT) with ^{123}I-iodobenzamide (IBZM), specifically binding to the cerebral dopamine receptor (D2), showed a decreased ganglia:cortex ratio, indicating a postsynaptic disorder. Nevertheless, biperidine, levodopa and carbidopa, and pergolide were prescribed, resulting in gradual but impressive clinical improvement within several weeks.

The clinical syndrome was unclear during the first few days after admission, but gradually developed into a hypokinetic rigid syndrome with resting tremor, generalised bradykinesia, and rigidity. This responded well to treatment with levodopa/carbidopa and a dopamine agonist.

Possible causes of a rapidly progressive form of parkinsonism are encephalitis, intoxication, head trauma, tumour, ischaemia, or hydrocephalus.[1] Imaging studies showed no abnormalities, thereby excluding the last three possible causes. Repeated history taking failed to disclose head trauma. The profession of the patient might suggest poisoning, but blood concentrations of manganese and copper were normal. The CSF showed mild pleiocytosis, but serological testing for various specific microorganisms did not show any recent infection. Radionuclide imaging showed a pattern similar to that seen in progressive supranuclear palsy or multiple system atrophy.[2]

The sequence of events strongly suggests a relation between the vaccination and the neurological syndrome, although the causal nature is difficult to prove.[3] To our knowledge, there are no reports of parkinsonism after tetanus immunisation. Alves *et al* reported a 5 year old boy who developed a postencephalitic rigid akinetic syndrome 15 days after vaccination for measles with live attenuated virus. Again, cause and effect remained open for debate.[4 5]

The tetanus vaccine used in our patient does not contain any living microorganisms. However, repeated injections with the tetanus toxoid might have caused hypersensitivity, and also an immunological cross reaction of antibodies with neuronal tissue directly after the last injection. This might also explain the pleiocytosis and raised protein and IgG content in CSF. The alternative explanation is that one of the substances in the vaccine vehicle, thiomersal or aluminiumphosphate, had a neurotoxic effect.

Although we are aware that a causal relation between the vaccine and the hypokinetic rigid syndrome is far from established, we have no better explanation. We wish to record the patient history as a reference, in case analogous patients might be seen in the future.

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Cardiogenic syncope in temporal lobe epileptic seizures

Cardiac arrhythmias may cause syncopal attacks masquerading as epilepsy. Conversely, epileptic seizures can induce tachyarrhythmias or bradyarrhythmias (and rarely, as a result, fainting). Differentiating between these two possibilities may prove difficult without concomitant ECG and EEG recording.

A 39 year old male lorry driver, without cardiac and neurological disorders and not taking medication, was admitted to a coronary care unit after a cluster of episodes of loss of consciousness preceded by epigastric warm sensation and a bitter taste in the mouth, and followed by pallor, sweating, muscle jerking, and rigidity with arrest of the pulse. The episodes occurred both in orthostatism and clinostatism. Clinical investigation, laboratory tests, clinostatic and orthostatic blood pressures, echocardiography, and ECG at rest, during exercise, and during carotid sinus massage were normal.

He experienced another attack while on continuous ECG monitoring. A nurse stated that the patient complained, while standing, of epigastric discomfort, followed by a fleeting phase of unresponsiveness and purposeless arm and mouth movements. A few seconds later he fell and showed a generalised tonic convulsion. The pulse, apparently normal during the initial phase of the episode, abruptly ceased when the patient collapsed. The ECG recording (fig 1) showed a progressive decrease of heart rate, culminating in a sinus arrest of 9.5 seconds, preceding the fall. Another four episodes of sinus arrest of 4-5 seconds, without tonic convulsions, were recorded on the next day.

A permanent demand ventricular pacemaker programmed to trigger at 40 beats per minute (bpm) was applied, but episodes of epigastric discomfort with lack of responsiveness and automatisms, not followed by syncope or convulsions, recurred on the next day and prompted his transfer to a neurological unit. A standard EEG showed focal spike and wave with delta slowing on the right centrotemporal region (C4-T4). A Medilog 9000 ambulatory EEG-ECG recording captured a seizure (fig 2) beginning with right centrotemporal recruiting sharp waves, followed by a progressive sinus bradycardia and culminating in a junctional escape rhythm at 47 bpm. The sinus rhythm was recaptured by a transient sinus tachycardia. Other episodes of psychomotor type occurred in subsequent days, but there were no falls or tonic convulsions. The epileptic seizures were secondary to a right anterior temporal low grade astrocytoma, and subsided after treatment with carbamazepine and removal of the mass. The pacemaker was left in place, and the patient has been free of seizures, without anticonvulsants, for three years.

Epileptic seizures often cause disturbances in cardiac rhythm, generally consisting of mild changes in heart rate such as sinus tachycardia. The possibility of life threatening cardiac arrhythmias has been suggested by the higher incidence of sudden unexpected deaths among patients with epilepsy than in the normal population.[1] Males, young adults with anatomical causes of seizure disorder, patients not receiving or receiving subtherapeutic levels of antiepileptic medication, with concomitant heart disease, and with alcohol misuse present the major risks.[1] Generalised tonic-clonic seizures, alone or in combination

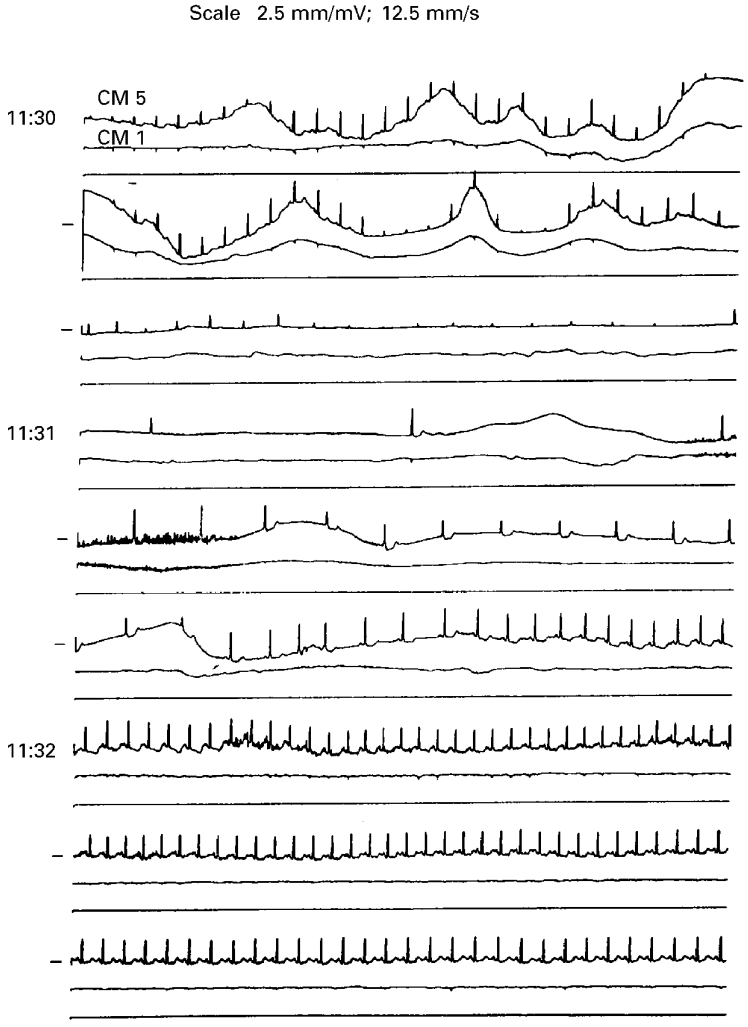

Figure 1 Two lead Holter recording of a syncopal attack (three uninterrupted minutes). During nausea and epigastric discomfort (minute 11.30) the sinus rhythm, superimposed by sweating and movement artifacts, progressively slows down. The syncope (minute 11.31) coincides with a sinus arrest of 9.5 seconds, intermingled with sporadic junctional escape beats and followed by a sinus rhythm (end of minute 11.31), that progressively accelerates up to 110 bpm (minute 11.32). Muscle artifact at the end of asystole indicates the occurrence of a tonic spell. CM1 = V1, CM5 = V5.

with partial complex seizures, are usually involved, and among proposed mechanisms is an intense sympathetic discharge to the heart, possibly time locked with vagal impulses and resulting in disordered cardiac rate, rhythm, or output.[1] There are only a few proved observations of epileptic bradycardia and asystole, and only in isolated cases[2–4] has the cardioinhibitory effect of a seizure been documented by simultaneous EEG and ECG.

In animals, neuromediated bradycardia has been elicited by electrical stimulation of various regions of the limbic system. In humans, repeated observations pioneered by Van Buren[5] have shown that seizure related bradyarrhythmias accompany electrical discharges originating from the temporal lobe, strengthening the hypothesis that neural structures within or adjacent to this lobe mediate cardioinhibition. A right-left hemispheric asymmetry for heart innervation has been suggested, but in the reported cases of ictal cardiac arrest right sided, left sided, and bilateral epileptic foci can be found.

Our case resembles that described by Smaje *et al*,[6] in which temporal lobe seizures secondary to a right hemispheric intracranial tumour induced recurrent episodes of sinus arrest, followed by syncope and muscle jerking; in this patient as well, surgical removal of the tumour reversed the epileptic seizures and the secondary cardiac involvement. Patients 1 and 2 of Constantin *et al* are similar.[7] In these patients, monitoring led to the false diagnosis of primary cardiac arrest and to the implantation of a permanent pacemaker.

In our patient, the presence of epigastric sensations and purposeless arm and mouth movements preceding the fall should have suggested the diagnosis of partial complex seizures, but clinical clues were disregarded in the face of a repeated documentation of sinus arrest. Actually, only simultaneous EEG and ECG recording makes it possible to recognise the concurrence and the timing of cerebral and cardiac disturbances, and this examination should be recommended in patients with episodes of loss of consciousness of an unclear nature. Finally, it is likely that falling and tonic convulsions after sinus arrest were anoxic rather than epileptic in origin, as they did not show when sinus arrest was shorter than nine seconds, nor after