the right side when the patient intended to open his mouth but on closing his mouth, no activation of the masseter and temporal muscles could be detected (figure). On the left side, the innervation pattern was normal. Routine needle EMG analysis of the right temporal, masseter, and anterior digastric muscles showed a chronic neurogenic pattern.

With the EMG needle in place we injected 75 u botulinum toxin (BOTOX©, Allergan) into the right masseter and temporal muscles. Three weeks after the injection the patient became progressively able to open his mouth, to eat, and to perform mouth care.

The pathogenesis of paradoxical activity of jaw muscles remains uncertain. For this patient we suggest that after a nuclear or axonal trigeminal lesion aberrant regeneration of trigeminal nerve fibres originally supplying jaw opening muscles led to false reinnervation of jaw closers. Thus, while intending to open the mouth, jaw closing muscles were falsely coactivated and prevented sufficient jaw opening. This innervation pattern was confirmed by EMG polygraphy. The interval of four months between infarction and the beginning of paradoxical activity of jaw muscles may reflect the time required for the perinuclear and intranuclear regeneration process.

Paradoxical activity of jaw muscles has to be differentiated from other clinically similar disorders. These include hemimasticatory spasm,[3] focal dystonia of jaw closing muscles,[4] local tetanus,[5] and diseases of the temporomandibular joint. Hemimasticatory spasm produces involuntary jaw closure due to unilateral contraction of jaw closing muscles, whereas paradoxical activity of jaw muscles occurs only during intended jaw opening. Jaw closure dystonia, which may occur unilaterally, is often linked to specific tasks or actions, may be accompanied by other dystonic movements, and lacks neurogenic change on needle EMG. Local tetanus can be identified by the absence of a silent period and its permanent muscle activity. Disorders of the temporomandibular joint would be obvious on radiological examination.

Botulinum toxin treatment is recommended as a simple, safe, and effective method in the management of paradoxical activity of jaw muscles.

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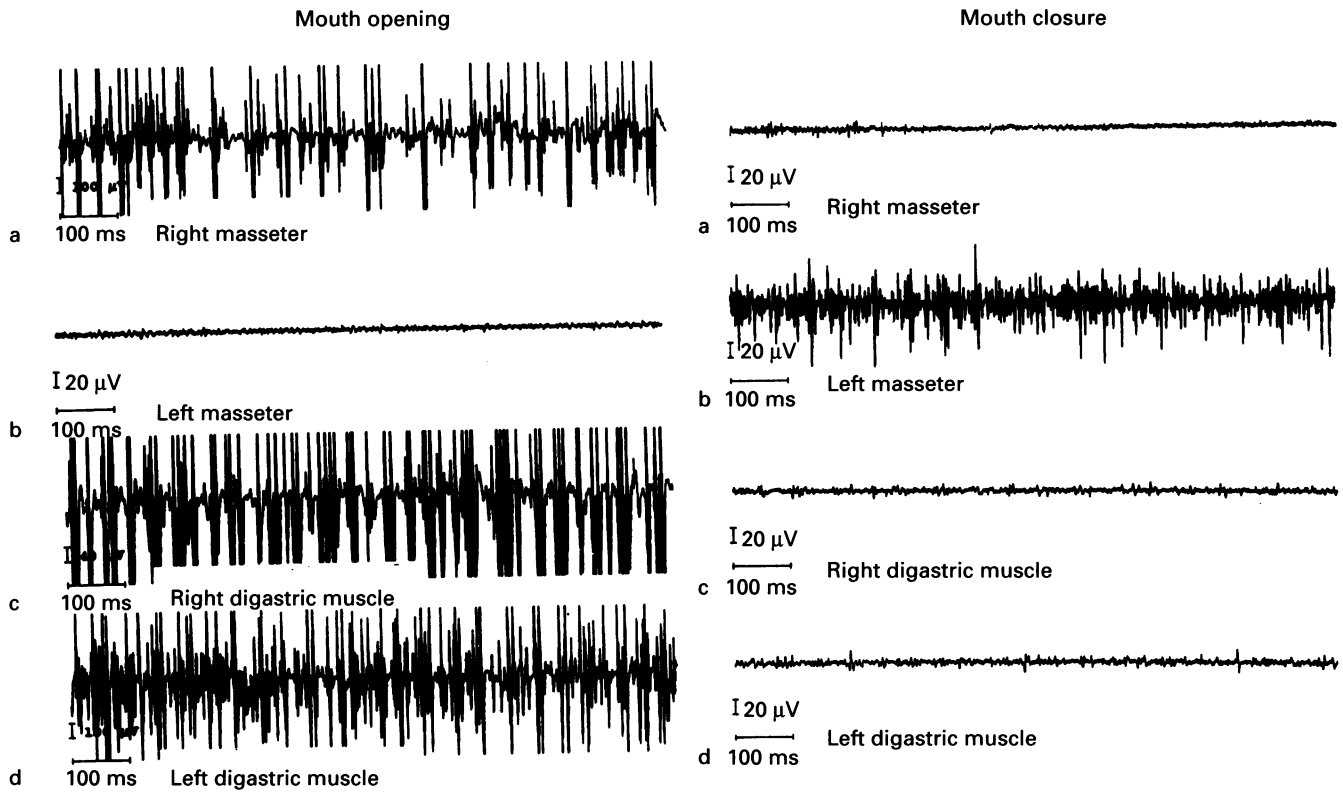

EMG polygraphy in paradoxical activity of jaw muscles using concentric needle electrodes. (A (a-d)) Paradoxical activation of the right masseter and normal activation of both digastric muscles when the patient intends to open his mouth. (B (a-d)) Lack of activation of the right masseter during mouth closure.

Treatment of severe tetanus by continuous intrathecal infusion of baclofen

A 72 year old woman with dysphagia and stiffness of the shoulders and the neck was diagnosed as having tetanus from EMG and clinical symptoms. We were not able to identify a wound as the likely source of infection. Her vaccination history was unclear, but she had not received any booster injection in the past 10 years. Treatment was started after two days and after a further two days she developed generalised, spontaneous, and provoked tonic muscle contractions leading to trismus, opisthotonus, risus sardonicus, and acute respiratory failure. We immediately started symptomatic treatment with mechanical ventilation and continuous midazolam and vecuronium. In addition to sedation and neuromuscular blockade antihypertensive treatment with urapidil and β-receptor blocking agents was necessary between days 2 and 7 and from day 19 until discharge from the intensive care unit. Specific intravenous treatment against causative *Clostridium tetani* consisted of penicillin G (20 million u/day) and human antitetanus immunoglobulin (10 000 U/day) on days 1 to 5. Antitetanus immunoglobulin was also given via the intrathecal route on days 15 and 16 (2000 U/day).

Complete resolution of the tonic muscle

spasms was not achievable despite very large doses of benzodiazepines and vecuronium. After informed consent from relatives, we began intrathecal baclofen[1] via a 32 gauge spinal catheter by lumbar puncture on day 13 and an infusion pump. Within the first 12 hours of infusion the neurological symptoms of tetanus gradually disappeared. On day 14 the daily amounts of midazolam and vecuronium were reduced and from day 15 benzodiazepines and muscle relaxants were no longer necessary. Baclofen alone was effective in abolishing the spasticity in a dose range of 750 to 1500 μg/day. Rigidity and spasms of the muscles of the trunk and the extremities ceased although the facial muscles still had a moderate degree of inducible contractures. On days 14, 15, and 16 repetitive bolus injections each of 1 mg of atropine sulphate were necessary to treat bradycardia.

Because the patient did not recover consciousness after stopping sedative medication, 2 mg flumazenil was given as an intravenous slow bolus injection on day 16 and caused an immediate transient awakening and return of severe muscle spasms. Baclofen was eventually stopped on day 28. During the next five days muscular tonus was moderately increased but the patient was free of pain and she was gradually weaned from mechanical ventilation.

In our case the initial dose of 850 μg baclofen per day given as continuous intrathecal infusion stopped muscle spasms within 12 hours even after withdrawal of all sedative and paralysing drugs on day 14. The patient remained unconscious, probably due to accumulation of midazolam. On the other hand central nervous effects of baclofen might also have contributed to the comatose state.[2] The central effects of baclofen are potentiated by benzodiazepines and are attributed to its action on GABA receptors in the brainstem and in the hippocampus. There are reports about antagonising the supraspinal side effects of baclofen by flumazenil while preserving its spinal action.[3] Our patient immediately regained consciousness after a dose of 2 mg flumazenil but she also exhibited all symptoms of severe tetanus. This finding might be indicative for a possible flumazenil counteraction of the spinal effects of baclofen. There is no doubt about the spinal action of baclofen on muscle spasms because of rapid cessation of symptoms after starting intrathecal infusion and because of recurrence of symptoms when baclofen treatment was interrupted on day 22. Baclofen injected into the lumbar subdural space spreads out cranially losing its efficacy by dilution within the CSF.[4] We observed in our patient remaining, although decreased, spasticity of the facial muscles and dose dependent muscle hypotonia of the limbs and the trunk after withdrawal of sedatives and relaxants, corresponding to the pharmacokinetics of intrathecal baclofen.

Intrathecal baclofen is an effective treatment of muscular paroxysms and contractions because it avoids disadvantages of conventional sedation and muscle relaxation (for example, prolonged coma, immobilisation). It is, however, a symptomatic treatment and does not shorten the duration of the disease.

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Ceftriaxone treatment of penicillin resistant neurosyphilis in alcoholic patients

Intellectual deterioration in alcoholic patients is often due to Korsakoff's syndrome, but alcoholism also carries increased risks of hepatic encephalopathy, pellagra, pontine or extrapontine myelinolysis, and traumatic brain lesions. The existence of alcohol dementia as a clinical entity remains controversial as a pathological basis is lacking. Neurosyphilis (inadequately treated) must be kept in mind in the differential diagnosis of Korsakoff's syndrome or so called "alcohol dementia".

We present the results of ceftriaxone treatment on two alcoholic patients who seemed to have been treated adequately for neurosyphilis, but who had dementia later.

Patient 1 was a 56 year old man with a history of alcohol abuse of several decades. He was admitted, having been found wandering in a neglected condition. He lived alone and was unemployed. In the past he had worked as an electrician and as an innkeeper. On admission the patient was apathetic and showed no insight. He was disoriented and had impaired memory functions. His mood was neutral, but soon became euphoric with grandiose delusions. Physical examination showed absent tendon reflexes in the legs. Serology and CSF tests showed evidence of syphilis of the CNS (table). Intravenous penicillin treatment was started, but could not be completed because of confusion and agitation. He was then treated with intramuscular procaine benzylpenicillin (2·4 MU twice daily for four weeks). This resulted in good clinical improvement, and at discharge after six weeks only minor disturbances in memory remained. We concluded that treatment had been adequate. The venereal disease research laboratory (VDRL) test became negative in serum but remained positive in CSF. The cell count, total protein concentration, and immunoglobulin fractions in CSF were decreasing.

One day after discharge the patient returned in a very frightened state. From that day his mental condition deteriorated, resulting in disorientation, progressive memory deficits, and depressive and psychotic symptoms. Neurological examination showed frontal and extrapyramidal symptoms. An EEG and MRI were normal. Tests for HIV were negative and serum concentrations of vitamins B1 and B12 were normal. SPECT showed signs of frontal hypometabolism. Eventually the patient was completely disabled and dependent on nursing. The differential diagnoses comprised Wernicke-Korsakoff's syndrome, primary degenerative dementia of the frontal type, and "alcohol dementia". Renewed activity of neurosyphilis seemed unlikely because serological tests remained unchanged. Nevertheless, the patient was treated with intramuscular ceftriaxone (1000 mg once daily for 14 days). This resulted in a good recovery and he eventually reached his former level of functioning (table). His CSF remained VDRL positive. The patient was discharged five months after treatment was started, having made a good recovery. Currently he lives on his own and has a paid full time job as a receptionist in a home for elderly people.

Patient 1: laboratory and psychometric data

	Before penicillin	*After penicillin Before ceftriaxone*	*After ceftriaxone*		
		Laboratory data			
Date	5 February 1990	23 April 1990	8 October 1990	8 April 1991	
Serum:					
VDRL	1 : 8	neg.		1 : 8	
TPHA	1 : 20480	1 : 40960		1 : 40960	
FTA-abs	positive	positive		positive	
CSF:					
VDRL	1 : 8	1 : 8	1 : 2	1 : 4	
TPHA	1 : 2048	1 : 32768	1 : 512	1 : 2560	
FTA-abs	positive	positive	positive	positive	
White cells/ml	333	81	27	27	
Protein (g/l)	0·56	0·46	0·42	0·34	
		Psychometric data			
Date	January 1990	August 1990	November 1990	April 1991	May 1992
IQ[1]	81	93	Cognitive functioning seriously retarded, testing not possible	—	106
MMSE[2] (score 0–30)	17	21		25	—
Memory[3] (deciles)					
Immediate recall	1	1		3	3
Delayed recall (after 20 minutes)	1	3		3	5