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

Patient 2 was a 72 year old man who had been admitted 10 times in the previous two years for alcohol detoxification. He had had untreated syphilis during the second world war. In 1966 he had been treated for dementia paralytica twice with penicillin-bismuth and once with chlortetracyclin. At that time he had recovered clinically but he did not become seronegative. In 1968 treatment had therefore been repeated. A decision to reinstate treatment had been taken in 1982, not because of any recurring symptoms or signs, but because of changing opinions about the adequacy of treatment. Treatment then consisted of benzathine benzylpenicillin (2·4 MU once a week for six weeks). He was already misusing alcohol. On one admission to our hospital for detoxification psychological testing showed only slight impairment of cognitive functions. One year later he was readmitted and found to be demented. His memory was seriously impaired, affect was shallow, and there was loss of decorum and social ability. The Korsakoff syndrome and primary degenerative dementia were considered as the most likely diagnoses. As both serum and CSF were VDRL negative and only weakly positive in *Treponema pallidum* haemagglutination (TPHA) and fluorescent treponemal antibody absorption (FTA-abs) tests, reactivation of neurosyphilis seemed unlikely. Because of our experience with patient A, however, patient B was also treated with ceftriaxone. Cognitive functions recovered to normal.

Both patients had had neurosyphilis and had been treated with penicillin intramuscularly. After initial recovery this treatment was considered sufficient; intravenous treatment with high dose penicillin for neurosyphilis is nowadays advocated to supply a continuous treponemicidal concentration in CSF.[4] In earlier years many patients received intramuscular penicillin. Serological tests had not shown relapse of neurosyphilis in our patients, but it should be borne in mind that the clinical significance of VDRL seroconversion after treatment is unknown, and that serological tests are often overvalued in determining the response to treatment of neurosyphilis.[5] The TPHA and FTA-abs are highly specific and sensitive for *Treponema pallidum* infection. These tests do not, however, give any information on clinical activity or time of infection, and are only useful in excluding the diagnosis of syphilis. VDRL tests are neither specific nor sensitive. Only strongly positive responses (< 1:16) signify active infection. It is therefore extremely difficult to prove whether neurosyphilis is active or not after an initial infection. In fact, the only argument that dementia was due to neurosyphilis in these two cases was the dramatic clinical improvement after treatment with ceftriaxone.

These cases draw attention to neurosyphilis as a differential diagnosis of (Wernicke-)Korsakoff's syndrome and as a possible cause of dementia in alcoholic patients. Both patients seemed to have been treated adequately with penicillin, but became demented after some time. In such cases treatment with ceftriaxone may lead to full recovery.

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Absence of REM sleep and altered non-REM sleep caused by a haematoma in the pontine tegmentum

The pronounced reduction or absence of REM sleep in patients with lesions of the pontine tegmentum indicates that this structure is implicated in generation of REM sleep in humans. This conclusion is based, however, on reports of a few patients with relatively large brainstem lesions.[1-3] Furthermore, information on the role of REM sleep in behaviour and memory consolidation is lacking.

Our patient had no REM sleep and abnormal Non-REM sleep caused by a haematoma confined to the tegmentum of the upper pons.

A 69 year old man had sudden instability and difficulty in speaking. He had a four year history of hypertension secondary to chronic renal failure. There was no history of alcohol consumption, sleep disturbances, or psychopathology.

On admission, neurological examination showed bilateral normoreactive miotic pupils, bilateral peripheral facial palsy, supranuclear horizontal gaze palsy, and cerebellar ataxia. He received nifedipine (60 mg daily) and three haemodialysis sessions weekly. After the stroke, the patient complained of persistent insomnia. No psychotropic, hypnotic, or sedative drugs were given.

Cranial MRI seven days after the first symptom showed mild cortical atrophy and a hyperintense signal in the upper pontine tegmentum in T1 and T2 weighted images (fig 1). Three polysomnograms (PSGs) were recorded. The first was over eight hours on the night of the 15th inpatient day. The second PSG was over 24 hours on the 30th inpatient day and the third four months later. The first and third PSGs were recorded in the sleep laboratory. The 24 hour PSG was recorded with an Oxford Ambulatory System at the inpatient's bed to avoid interference from laboratory conditions.

Parameters of PSG recorded were EEG (C3-A2), EMG, right and left electro-oculogram (R-EOG, L-EOG), left leg movements (LM), body position (BP), respiratory movements (RESP), and EEG (Fp1-C3, C3-T3, T3-O1, Fp2-C4, C4-T4, T4-O2).

Electrodes to record eye movements were placed 1 cm lateral and 1 cm above the outer canthus of each eye. Both electrodes were referred to the right ear.

No psychotropic, hypnotic, or sedative drugs were given on the days before or during PSG recording days.

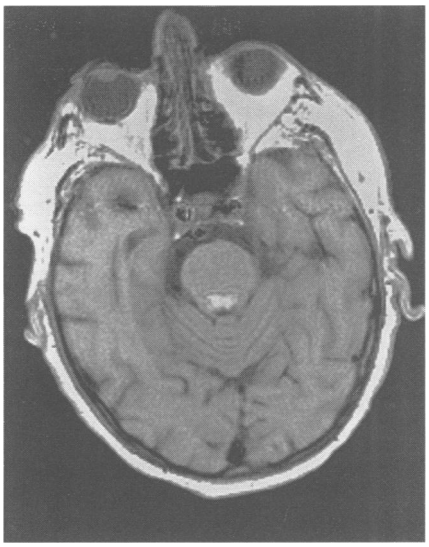

Figure 1 Increased signal on T1 weighted MRI (haematoma) in the pontine tegmentum.

Three different PSG patterns were recorded: (*a*) wake time with occipital alpha rhythm and a few vertical eye movements;. (*b*) a decrease of over 50% in alpha rhythm and moderate decrease of EMG activity; (*c*) a slow wave pattern with more than 20% delta activity and a greater decrease of EMG activity. (fig 2).

The first PSG recording, performed on the 15th inpatient night, showed severe insomnia with absence of REM sleep, absence of beta spindles and K complexes, and scarcity of deep slow sleep. There was a pronounced decrease in the total sleep time (TST) of three hours and 56 minutes. A 24 hour PSG performed 15 days later showed the same characteristics and TST was three hours and 51 minutes (fig 2). The most recent PSG, recorded four months later, showed persistence of the same sleep disorder.

Neurological examination during the three PSG sessions remained unchanged. After 18 months of follow up, mild insomnia (four to five hours of sleep nightly), supranuclear horizontal gaze palsy, bilateral peripheral facial palsy, and instability persisted.

During the follow up period, no psychic disturbances were found. The neuropsychological studies during this time (one month, four months, 12 months, and 18 months), including auditory verbal learning test (Rey), long term retention memory test, President's test, and memory span for digits (WAIS), showed no learning disability and remote, recent, and immediate memories were normal.

Previous cases of absence of REM sleep in humans have been associated with large lesions in the pons with bilateral damage of the tegmentum, mostly in patients with pronounced neurological deficits.[1-3] The well delineated lesion in our patient confirms that REM sleep features originate in the pontine tegmentum. In agreement with other reports the coexistence of severe insomnia and supranuclear horizontal gaze palsy suggests that REM sleep features generate in areas closely related to control of horizontal eye movements.[1 3]