Another finding in our patient was the absence of sleep spindles. As emphasised in previous reports, lesions of the pontine tegmentum may involve the peduncle-pontine ascending connections of the nucleus reticularis thalami and provoke the absence of beta spindles and K complexes.\(^1,2\)

It is important to perform sleep recording over at least 24 hours to determine the exact sleep time and to rule out disorders of circadian rhythm, especially in patients in hospital with severe physical disability.\(^3\) In our patient, the circadian rhythm was stable with predominance of wake time during the day and sleep periods during the night, even in the inpatient period.

The psychic effects of REM sleep deprivation are not well known, partly because it is extremely difficult to produce long periods of complete REM deprivation in humans. It has been suspected that REM sleep is important for normal behaviour and that severe REM deprivation might cause irritability, hallucinations, delusions, obsessions, and schizophrenia.\(^4\) This conclusion is not supported by recent studies\(^2\) or our case.

Finally, the hypothesis that REM sleep is essential for memory consolidation is also of interest.\(^5\) To our knowledge, this is the first case of total absence of REM sleep in which neuropsychological studies have been performed, and there were no abnormal memory consequences.

This work was presented in abstract form to the XLV Annual Meeting of the Spanish Neurological Society and to the 12th Congress of the European Sleep Research Society.

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There have been a few reports of cluster headache with pain on one side and autonomic features on the other; sometimes the patient has previously had pain on the side of the previous autonomic symptoms. Experiments with balloon catheters have shown that pain around and above the eye can be produced by stretching the internal carotid artery just below the syphon, a site where the artery is surrounded by the autonomic fibres supplying the eye. A sterile inflammatory response in the vessel wall might simultaneously narrow the lumen and press the nerve plexus against the bony skull, thus producing the watering eye and Horner’s syndrome. Dilatation of the ipsilateral ophthalmic artery, without any changes in the internal carotid artery or circle of Willis, was seen during MRI in a patient who did not have a Horner’s syndrome.

The explanation for the apparent lack of correlation of the severity of the inflammatory process (as judged by the pain) with the severity of the autonomic dysfunction in this patient remains obscure.

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Optic neuritis in cerebral toxocariasis

Optic neuritis occurs in an isolated manner or in the presence of multiple sclerosis. Rarely optic neuritis has been described in association with a nematode infection. All of the documented lesions to date involve the optic nerve head, resulting from a direct intracocular infection. This article presents the first demonstration of retrolubar optic neuritis verified by MRI in a patient with esophagic meningocerebralmyelitis due to *Toxocara canis*.

A 21 year old woman was admitted to our hospital because of headache, low grade fever, and convulsions. Her illness had begun four weeks before admission with a constriciting frontal headache and fever of 37-5°C, followed by several episodes of convulsions two weeks later. She had been exposed to a dog for eight years, since it was a puppy. General physical examination on admission showed a temperature of 37°C but no evidence of skin rash or hepatomegaly. The mental state of the patient was slightly impaired. Positive findings on neurological examination included evidence of meningeal irritation such as neck stiffness and Kernig’s sign. Cerebellar ataxia, with a predilection for the right side, was also noted. Complete ophthalmological examination, including determination of visual acuity, the anterior segment of the eye, and retinal, yielded normal results.

Laboratory studies showed a slight leukocytosis (9100/μl) with an increased number of eosinophils (23-6%). Her CSF contained 330 leucocytes/μl with 30% of eosinophils, and a protein concentration of 55 mg/dl; Synthesis of IgG was increased at 43-6 mg/day. Cultures of blood, urine, and CSF, and multiple examinations of stool for ova and parasites were all negative. Results of CT and MRI (1-5T unit) on admission were unremarkable. Indirect immunofluorescence tests, with enzymo-conjugated *Toxocara canis* egg, were positive for both serum and CSF. We also performed an immunoblotting assay (IBA) and an enzyme linked immunosorbent assay (ELISA) with the secretory products of second stage larvae of *Toxoocara canis* as an antigen according to methods described previously. Both the IBA and ELISA yielded positive results. The ELISA values for *Toxocara canis* in this patient were 1-687 in serum and 0-049 in CSF, whereas those in controls were 1-060 (0-340) (mean, SD) and 0-025 (0-001) (n=10), respectively. By contrast, ELISA tests for antibodies to Angiostrongylus cantoneis, *Ascaris*, *Dipylidium caninum*, *Spirometra erinacei*, and *Trichinella spiralis* were negative.

Despite treatment with diethylcarbamazine (300 mg/day for eight weeks) and prednisolone (40 mg/day), leg spasticity, sensory impairment below the level of C4, and Lhemitte’s sign developed. MRI performed four weeks after admission showed lesions located mainly in cortical or subcortical layers of cerebrum, the cerebellum, and the upper cervical spinal cord. These lesions had a hyperintense appearance on T2 weighted images and were enhanced with gadolinium.

Ten weeks after admission, the patient began to complain of blurred vision in the upper visual field of the right eye and pain behind the affected eye with attempted eye movement. An ophthalmological examination showed a reduced visual acuity of light perception OD, a right relative afferent pupillary defect, and a normal optic disc, indicating retrolubar optic neuritis. Repeat examination of her CSF showed 19 leucocytes/μl without eosinophils, 52 mg/dl protein, and IgG synthesis of 7-5 mg/day. In addition, the occurrence of the right frontal lobe lesion, CT and MRI disclosed swelling and a gadolinium enhanced lesion of the right optic nerve (figure) respectively. Brain biopsy of the frontal lobe lesion failed to find the worm but showed the accumulation of inflammatory cells around the vessels. Treatment with intravenous methylprednisolone (1000 mg for three days) and cyclosporin (4 mg/kg/twice daily) failed to lead to the recovery of her visual acuity. Two weeks later, mild oedema of the optic disc with minor hyperaemia became evident in the right eye. Regardless of several sub-Tenon’s betamethasone injections (5 mg) at this stage, the patient developed optic atrophy of the right eye and her visual acuity remained reduced at 20/200 OD.

Twelve weeks after the initial attack of optic neuritis, the patient developed retrolubar pain and loss of vision in her opposite left eye. Swelling and a gadolinium enhanced lesion was again evident in the left optic nerve with MRI, whereas a pronounced reduction was noted in size and number of the lesions in the other areas. After the intensive treatment with intravenous methylprednisolone and sub-Tenon’s betamethasone, the ocular pain rapidly subsided, but her visual acuity was reduced to 20/200 OS. A repeat assay for *Toxocara canis* antibody showed a decrease in titre to 0-739 in serum and 0-032 in CSF. The immunosuppressive drugs were gradually discontinued. Although neurological sequelae, such as diminished visual acuity and cerebellar ataxia, have remained, her neurological follow up during the past year has been unremarkable, and she is not taking medication.

This is the first report of optic neuritis

MRI of the optic nerves. In addition to the right frontal lobe lesion, T1 weighted MRI images (TE 440 ms, TR 15 ms) show pronounced gadolinium enhancement of the orbital segment of the right optic nerve (arrow).
Recurrence of cluster headaches presenting with a virtually painless Horner's syndrome.

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