improvement in neurological symptoms, indicating that the cytomegalovirus infection was a cause of the neurological disease. The etiology of Bickerstaff's brainstem encephalitis is still unclear. A relation with herpes simplex virus infection has been noted, but no patients with Bickerstaff's brainstem encephalitis associated with cytomegalovirus infection have been reported.

With regard to the pathogenesis of Bickerstaff's brainstem encephalitis, an immune mechanism has been considered. In this patient, the presence of serum anti-GQ1b antibody, which is common in Fisher's syndrome, indicated that humoral autoimmune mechanisms, common to Fisher's syndrome, function in the development of Bickerstaff's brainstem encephalitis. The typical signs of meningoencephalitis—namely, fever at the onset of neurotic symptoms, meningeal irritation, and CSF pleocytosis—and detection of cytomegalovirus DNA in the CSF may indicate the involvement of cytomegalovirus infection. Both cytomegalovirus infection and a post-infection autoimmune mechanism may have caused clinical symptoms in this patient.

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Raymond syndrome (alternating abducens hemiplegia) caused by a small haematoma at the medial pontomedullary junction

Raymond syndrome is characterised by ipsilateral abducens nerve palsy and contralateral hemiplegia. Pure Raymond syndrome is extremely rare, as many nuclei and fibres exist near the root fibres of the abducens nerve. This is the first report in which the precise localisation of a pure form of Raymond syndrome was determined by MRI.

A 39 year old man awoke with horizontal diplopia, especially on right lateral gaze. Five days later, a Hess chart examination performed by an ophthalmologist showed paresis of the right lateral rectus muscle. On admission 19 days after onset, the patient showed a mild paresis of the right abducens nerve and a subtle weakness of his left leg with moderate hyper-reflexia in the left upper and lower limbs. The Babinski reflex was positive and the abdominal reflex was absent on the left side and the Babinski reflex was negative and the abdominal reflex was positive on the right side. No facial weakness or deviation of the tongue on protrusion was found. All other general and neurological examinations were normal. Routine blood and urine examinations were normal. Evaluations of short latency somatosensory evoked potentials to posterior or thalial nerve stimulation, brainstem auditory evoked potentials, and blink reflex proved normal. Head CT was normal, but a brain MRI done 31 days after onset showed two punctate high signal intensity spots surrounded by low signal intensity areas at the right pontomedullary junction (arrows). This lesion was probably produced by a haemorrhage from a cavernous haemangioma at the pontomedullary junction.

Both Millard-Gubler syndrome (facial palsy and contralateral hemiplegia) and Raymond syndrome are well known to induce crossed paralysis due to a caudal pontine lesion. The pure form of either syndrome has, however, rarely been reported. The lesion producing the pure Millard-Gubler syndrome is located more laterally than seen in our patient, whereas that producing isolated abducens nerve palsy is located more dorsally. As the haemorrhage was restricted to the ventral and medial pons, our patient was considered to show pure Raymond syndrome.

Pupillary dilatation and arm weakness as negative ictal phenomena

Transient ictal hemiplegia is an uncommon feature of epileptic attacks that were classified by Gastaut and Broughton as unilateral atonic seizures. The present case was of particular interest because hemiplegia was accompanied by dilation of the pupil on the side of the hemiplegia. A boy aged 9 years had a history of episodic weakness of his left upper and lower limbs, sometimes preceded by a sensation like a dog averting his head since the age of 5. His mother said that he would stare and his left arm then dropped limply to his side while his left leg became weak for about 10 to 40 seconds. During this time his left pupil dilated. In some episodes his left eyelid fluttered and the left side of his mouth turned up and his left arm and leg remained weak. The attacks increased in frequency until he was having two to eight each day, but subsided to once daily when carbamazepine treatment was started. There was no history of head injury or other relevant illness and no family history of epilepsy. His EEG showed an almost continuous sharp and slow wave discharge arising in the right parietal region. Brain CT was normal but MRI four years later showed a hypointense area involving both grey and white matter in the right parietal lobe; there was no mass effect or evidence of blood products surrounding the lesion.

At the age of 13 he underwent cranio- and cerebral corticography, supported by the presence of an epileptic focus in the area surrounding an atrophic gyrus in his right parietal cortex. The abnormal area was then excised. The histology report (Dr W A Evans) concluded that "I found this lesion hard to classify. It is most likely a hamartoma, possibly of a similar nature to the focal dysplasia of the cerebral cortex described by Taylor et al." There was no postoperative neurological deficit and he was free of seizures until eight months later when his carbamazepine dosage was reduced from 1000 mg to 400 mg daily. Three years after this carbamazepine dose was again reduced, when he had a recurrence of daily attacks of fluttering of his left eyelid and weakness of his left arm, but not the left leg, lasting 20 seconds. His EEG showed focal right partial slow activity without epileptogenic features. Since then he has been subject to episodes about every 10 days with dilation of the left pupil, weakness of the left arm, and some twitching of the left side of his face lasting about 10-20 seconds. He has never had any jerking or involuntary movement of his left arm.

Contraction of the left pupil in association with hallucinations projected into the left visual field was reported as an ictal phenomenon by Lance and SME and