protein antibodies to detect exclusively normal or hyperphosphorylated tau protein may help to further evaluate the diagnostic value of tau determinations in CSF.

Moreover, these results support a recent study. Our investigation showed a highly significant increase of CSF total tau protein in patients with probable Alzheimer’s disease compared with elderly depressed patients, patients with other neurological disorders, and young and elderly controls. It seems plausible that these increases are the result of enhanced release of tau protein from damaged neurons into the extracellular space and, as a consequence, into CSF. Recent results suggested an arbitrary cut off value of 200 pg/ml, at which only a small percentage of healthy controls are positive. Based on our data, we would propose a 10% higher cut off value, of about 225 pg/ml, to better distinguish the elderly depressed patients as well as young and elderly controls. The observed difference between patients with Alzheimer’s disease and elderly depressed patients confirms a recent study and is of particular interest. Depressive periods can be accompanied by a severe impairment of attention, receptivity, and concentration which may mimic a dementing process. Thus early neurochemical identification of a depressive syndrome is of great importance for appropriate therapeutic strategies. The reason for more tau protein in depressed patients than in young controls is unknown. On the other hand, the non-significant difference between young and elderly controls leads to the assumption that increased tau protein is not merely a marker of aging itself.

There are contradictory reports regarding the correlation between MMSE and total tau protein immunoreactivity in the CSF of patients with Alzheimer’s disease. From our data there was a significant correlation only when including the whole range of scores, which became non-significant when excluding the patients with severe dementia. These results corroborate neuropathological findings which showed a significant correlation between the number of neurofibrillary tangles and MMSE only when including severely demented patients. In conclusion, total tau protein concentration in CSF is an important candidate for discriminating patients with Alzheimer’s disease from elderly depressed patients as well as from young and elderly controls without mental illness. Further studies on other neurodegenerative diseases are in progress. Moreover, different combinations of tau protein antibodies to detect exclusively normal or hyperphosphorylated tau protein may help to further evaluate the diagnostic value of tau determinations in CSF.

We report a hypertensive man with an isolated gaze disorder consisting of a severe bilateral abduction palsy and a subclinical bilateral gaze palsy—that is, bilateral conjugate saccadic palsy—with no other identifiable aetiology, who had a bilateral pontine infarction involving the rostral PPRF, seen on MRI.

A 64 year old man with a history of mild hypertension and hyperlipidaemia was referred for evaluation of painless horizontal diplopia of sudden onset. There was no history of head trauma, diabetes mellitus, or any other systemic disorder. Blood pressure was 150/100 mm Hg. Neurological examination was normal except for a complex type of gaze defect—namely, mild bilateral conjugate saccadic palsy of an additional severe right lateral rectus weakness and pronounced esotropia of the left eye on straight gaze. No nystagmus or internuclear ophthalmoplegia was seen and the rest of the extraocular movements were full. "Doll’s head" manoeuvre (vestibular ocular reflex) in the direction of the palsy was intact. Pupil sizes and light reflexes were normal in both eyes. Sensation in the first division of the trigeminal nerve was intact. Facial and mas- seter muscles were of normal strength and there were no signs of long tract involv- ment. Results of routine laboratory tests, including CSF analysis, were unremarkable. Brain MRI (0-5 T) showed only minor bilateral hyperintensities at the upper pontine tegmentum, without definite relevance. One week later he suddenly complained of dizzi- ness and a more intense diplopia on hori- zontal gaze. His neurological examination showed a newly developed complete paraly- sis of abduction of the left eye. A second MRI disclosed bilateral hyperintensities at the rostral pontine tegmentum compatible with infarction (figure). The oculomotor function remained resolved two months later with only a persistent mild bilateral saccadic palsy and no lateral rectus weakness.

Our patient’s brain MRI showed a bilateral infarction with involvement of the ros- tral pontine tegmentum containing the PPRF, the structure responsible for the gen-
eration of horizontal saccades. In animals, this is located in the nuclei reticularis pontis oralis (NRPO) at the level of the upper pons. The patient presented a clinically evident paralysis of abduction of both eyes with a subclinical bilateral gaze palsy. Lesions impinging only on the area of the NRPO, with no abnormal signals in other pontine sections affecting structures such as the low PPRF (nuclei reticularis pontis caudalis (NRPC)), abducens nucleus, or the abducens nerve fasciculus. Bronstein et al, in their extensive review of the abnormalities of horizontal gaze, reported three patients with a single brainstem episode of unilateral gaze palsy with an additional “mild” sixth nerve palsy to the side of the gaze palsy. They attributed weakness of the lateral rectus to involvement of the sixth nerve nucleus and concluded that the presence of this sign may be useful in identifying the site of the lesion in the mild gaze palsies. Cells lying in the PPRF project directly into the abducens nucleus. The connectivity of this network has been investigated in detail and shows that several different groups of saccade related premotor neurons exist, the most important of which are the excitatory burst neurons. We think that involvement of these burst neurons or their descending axons as they course towards the sixth nerve nucleus could have contributed to the findings in our patient. Selective lesions within the PPRF might affect horizontal gaze in different ways, including “sixth nerve palsies” as in the patient in this paper.

Pontine infarcts should always be considered in the differential diagnosis of isolated sixth nerve palsies and in more complex gaze disorders. So in these patients, precise examination including MRI is needed.

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Bilateral infarction of the rostral pontine tegmentum as a cause of isolated bilateral supranuclear sixth nerve palsy related to hypertension.

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