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Matters Arising

Combined gaze palsy of horizontal saccades and pursuit contralateral to a midbrain haemorrhage

Sir: Drs Bolling and Lavin[1] report that impairment of both contralateral saccades and smooth pursuit was due to a lesion affecting the ipsilateral midbrain reticular formation. However, damage to one midbrain reticular formation results in loss of all ipsilateral horizontal rapid eye movements, as well as smooth pursuit, preserving vestibular-induced ipsilateral movements.[2,3] In our opinion, the ocular findings in their report would suggest that the supranuclear pathway for voluntary saccades and smooth pursuit (occipito-parieto-pontine pathway) were affected rather than the midbrain reticular formation. Furthermore there is no evidence that these supranuclear pathways have the same location as the reticular formation in the midbrain.

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References

1 Bolling J, Lavin PJM. Combined gaze palsy of horizontal saccades and pursuit contralateral to a mid-brain haemorrhage. *J Neurol Neurosurg Psychiatry* 1987;**50**:789–91.
2 Deleu D, Solheid C, Michotte A, Ebinger G. Dissociated ipsilateral horizontal gaze palsy in one-and-a-half syndrome: a clinicopathological study. *Neurology* 1988;**38**:1278–80.
3 Pierrot-Deseilligny C, Chain F, Serdaru M, Gray F, Lhermitte F. The one-and-a-half syndrome. Electro-oculographic analysis of five cases with deductions about the physiological mechanisms of lateral gaze. *Brain* 1981;**104**:665–9.

Dr Lavin replies:

Dr Deleu implicates the midbrain reticular formation in the loss of voluntary ipsilateral horizontal eye movements (pursuit and saccades), citing two studies[1,2] in support. Both these studies describe patients with the "one-and-a-half syndrome" caused by lesions of the paramedium *pontine* reticular formation (PPRF). The subject of the first study, and to a lesser extent patient one in the second study, had some involvement of the lower midbrain; however, the brunt of disease affected the rostral pontine reticular formation in both patients.

That the supranuclear pathways for pursuit travel through the ipsilateral midbrain reticular formation in man[3,4] and monkeys[5] is well documented. Furthermore, thalamic injury has been associated with impaired ipsilateral pursuit in man.[6]

In our patient the lesion involved the rostral midbrain contralateral to the gaze palsy.[7] Without pathological verification one cannot say with absolute certainty that the thalamus was not involved; however, the patient had neither clinical nor radiological (CT and unpublished MRI) evidence of thalamic involvement.

The kernal of our report is that the supranuclear pathways for smooth pursuit eye movements pass through the contralateral midbrain and therefore must decussate twice, at least, in order to innervate the ipsilateral pontine nuclei. The apparent paradox of ipsilateral[3,5] and contralateral[7] pursuit deficits with unilateral midbrain lesions may be explained by the existence of parallel pathways. We stand by our original conclusions.

References

1 Deleu D, Solheid C, Michotte A, Ebinger G. Dissociated ipsilateral horizontal gaze palsy in one-and-a-half syndrome: A clinical pathological study. *Neurology* 1988;**38**:1278–82.
2 Pierrot-Deseilligny C, Chain F, Serdaru M, Gray F. Lhermitte F. The one-and-a-half syndrome. Electro-oculographic analysis of five cases with deductions about the physiological mechanisms of lateral gaze. *Brain* 1981;**104**:665–9.
3 Zackon DH, Sharpe JA. Midbrain horizontal gaze paresis. *Ann Neurol* 1984;**16**:495–504.
4 Bril V, Sharpe JA, Ashby P. Midbrain asterixis. *Ann Neurol* 1979;**6**:362–4.
5 Komatsuzaki A, Albert J, Harris HE, Cohen B. Effects of mesencephalic reticular formation lesions on optokinetic nystagmus. *Exp Neurol* 1972;**34**:522–34.
6 Brigell M, Babikian V, Goodwin JA. Hypometric saccades and low gain pursuit resulting from a thalamic lesion. *Ann Neurol* 1984;**15**:374–8.
7 Bolling J, Lavin PJM. Combined gaze palsy of horizontal saccades and pursuit contralateral to a midbrain hemorrhage. *J Neurol Neurosurg Psychiatry* 1987;**50**:789–91.

Multiple sclerosis: correlation of magnetic resonance imaging with cerebrospinal fluid findings

Sir: Honig *et al*[1] reported that magnetic resonance imaging (MRI) performed by them was normal in 55% of clinically definite multiple sclerosis (CDMS) of less than 2 years duration. In addition they found normal MRI in 26% of CDMS patients with supportive CSF changes. These figures suggest lower sensitivity of MRI than that recorded from other contemporary studies.[2]

One explanation they proposed for their findings is that other studies have looked predominantly at long established disease. However, when MRI was performed during the first presentation of acute idiopathic clinically isolated optic neuritis it was found by us that 64% had multiple brain lesions. This also corresponds well with epidemiological predictions of progression to CDMS, and using combined clinical and imaging evidence for relapse 56% of the same group developed CDMS within one year of follow up.[3]

Another of their proposed explanations was the occurrence of spinal without cerebral lesions. In our study of isolated spinal cord syndromes 75% had brain lesions indistinguishable from those seen in CDMS, also making this explanation unlikely.[4]

The reasons for their low rate of lesion detection possibly lie in their imaging protocol. Firstly 10 mm thick slices are suboptimal as small lesions may not be detected due to partial volume effects (some of our optic neuritis group were, however, scanned with 10 mm slices); MRI with most current systems will image adequately at 4 mm slice thickness and below. Secondly a 0·35T machine will have a lower signal-to-noise ratio than comparable higher field machines, resulting in noisier images and less certainty in lesion detection. Thirdly signal-to-noise can be improved by the design and optimisation of receiver coils and scanning sequences. We have found that inferior images may result from double as opposed to single echo sequences.

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References

1 Honig LS, Siddhartan R, Sharemata WA, Sheldon JJ, Sazant A. Multiple Sclerosis: correlation of magnetic resonance imaging with cerebrospinal fluid findings. *J Neurol Neurosurg Psychiatry* 1988;**51**:280.
2 Baumhefner RW, Tourtellotte WW, Ellison G, *et al. Neurology* 1986;**36(Suppl.1)**:283.