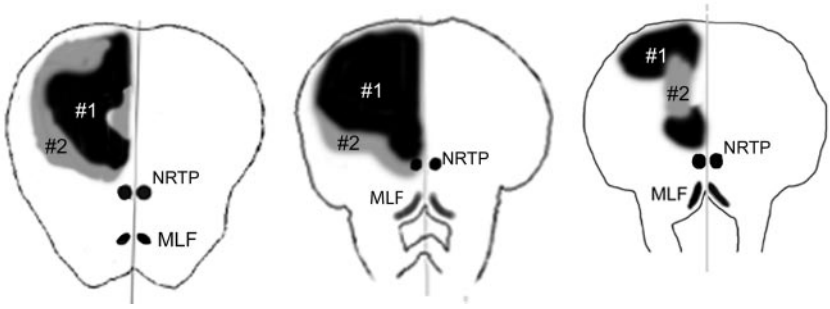
Vergence palsy in pontine lesion

Rambold et al. showed that not only midbrain but also pontine lesions cause a palsy of "fast" vergence eye movements. In the figure, the lesions involve the nucleus reticularis tegmenti pontis but spare the medial longitudinal fascicle, the omnipause neurons, and the midbrain.

see page 338



Throwing a glance: Fast vergence eye movements VIDEO

Commentary by R. John Leigh, MD

Most objects in our visual world lie in different directions *and* at different distances. Thus, during visual search, most rapid shifts of the fixation point require movements of the eyes in the same direction (saccades) *and* simultaneous adjustment of the angle between them (vergence) to land both eyes on target. Contrast this natural behavior with conventional testing of vergence at the bedside: the examiner slowly brings a visual target (such as his finger or a pencil tip) slowly towards the patient's nose, and the eyes should smoothly pursue the target. We may well ask: are we testing vergence incompletely and, if so, what are we missing?

In this issue of *Neurology*, Rambold et al. report two patients with pontine infarctions who showed a selective defect (slowing) of "fast" vergence when they were asked to "jump" their fixation point alternately between far and near targets; in one patient, slowing of "fast" vergence movements was evident clinically. In neither patient could the slowing of vergence be attribute to slow saccades. Both patients also showed a mild impairment of "slow" vergence, corresponding to smooth tracking of a target during clinical testing. In a prior report, Rambold et al.[1] reported two other patients with selective defect of "slow" vergence movements, but preserved "fast" vergence. Taken together, their findings point to separate substrates for different types of vergence response. Where were the lesions?

In all four patients, MRI demonstrated unilateral paramedian pontine infarction; more rostral lesions were associated with impaired "fast" vergence and more caudal lesions with impaired "slow" vergence. The notion of pontine lesions causing selective defects of vergence eye movements might seem surprising, since basic and clinical studies attest to the critical role of the dorsal midbrain in the control of vergence.[2] On the other hand, pontine nuclei, especially nucleus reticularis tegmenti pontis, have been shown, in monkey, to contain neurons that discharge for saccades, vergence, or smooth pursuit.[2,3] While neuroscientists work to elucidate the role of the pontine nuclei in the control of eye movements, these two clinical reports contribute to our knowledge of human vergence control. And they prompt clinicians to test vergence not just by "follow my finger," but by asking the patient to jump their fixation point alternately between far and near targets (see the video on the *Neurology* Web site at www.neurology.org).

References

1. Rambold H, Neumann G, Helmchen C. Vergence deficits in pontine lesions. Neurology 2004;62:1850–1853.
2. Leigh RJ, Zee DS. The neurology of eye movements. New York: Oxford University Press, 1999:286–318.
3. Gamlin PDR. Neural mechanisms for the control of vergence eye movements. Ann NY Acad Sci 2002;956: 264–272.

see page 338

Copyright © 2005 by AAN Enterprises, Inc.

Copyright © by AAN Enterprises, Inc. Unauthorized reproduction of this article is prohibited.

Neurology®

January 25 Highlight and Commentary: Throwing a glance: Fast vergence eye movements

R. John Leigh MD

Neurology 2005;64;179
DOI 10.1212/WNL.64.2.179

This information is current as of January 24, 2005

Updated Information & Services	including high resolution figures, can be found at: http://n.neurology.org/content/64/2/179.full
Supplementary Material	Supplementary material can be found at: http://n.neurology.org/content/suppl/2005/01/14/64.2.179.DC1 http://n.neurology.org/content/suppl/2005/01/18/64.2.179.DC2
References	This article cites 2 articles, 1 of which you can access for free at: http://n.neurology.org/content/64/2/179.full#ref-list-1
Permissions & Licensing	Information about reproducing this article in parts (figures,tables) or in its entirety can be found online at: http://www.neurology.org/about/about_the_journal#permissions
Reprints	Information about ordering reprints can be found online: http://n.neurology.org/subscribers/advertise

Neurology ® is the official journal of the American Academy of Neurology. Published continuously since 1951, it is now a weekly with 48 issues per year. Copyright . All rights reserved. Print ISSN: 0028-3878. Online ISSN: 1526-632X.

AMERICAN ACADEMY OF NEUROLOGY®