

# Optokinetic Asymmetry in Internuclear Ophthalmoplegia

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**The recognition of internuclear ophthalmoplegia in patients in whom there is full range of adduction rests on the "dysmetria test" and the "optokinetic test." Both the dysmetria present with rapid refixations and the optokinetic asymmetry are discussed in terms of the neurophysiologic mechanism behind the slow adducting saccades in internuclear ophthalmoplegia.**

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Internuclear ophthalmoplegia is a sign characterized by impairment of adduction in one eye and nystagmus of the opposite, abducting eye. It signifies a lesion in the medial longitudinal fasciculus between the sixth and third cranial nerve nuclei on the side in which adduction is lost. Such lesions result in varying degrees of limitation of adduction up to and including a total inability to cross the midline during attempted contralateral gaze. However, many patients with nystagmus of an abducting eye, suggestive of internuclear ophthalmoplegia, can fully adduct either eye. For such cases with full adduction, Smith and David<sup>1</sup> used two clinical tests designed to demonstrate a more subtle adduction impairment by requiring each medial rectus muscle to

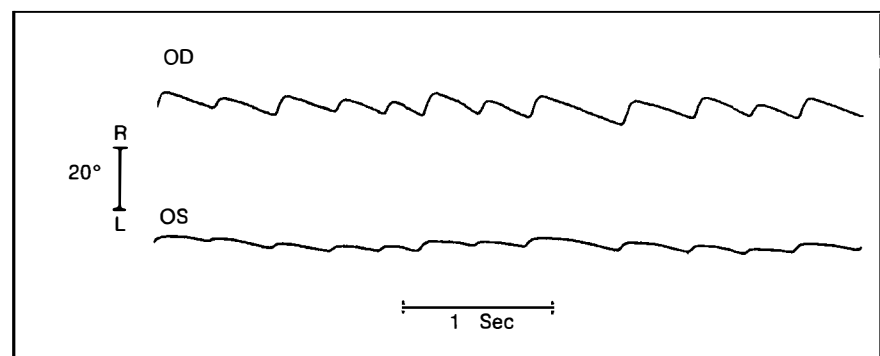
make saccadic eye movements. In the "dysmetria test," rapid refixations disclose a greatly slowed adduction saccade but a normally rapid abduction, usually with an overshoot. In the "optokinetic test," when the involved medial rectus is responsible for the fast phase of nystagmus, that eye has a smaller amplitude nystagmus response than the other eye.

These signs have become generally accepted for the recognition of internuclear ophthalmoplegia and are of particular value in those more subtle cases in which there is full range of adduction. We should like to suggest, in this brief report, an explanation for the observed optokinetic eye movements and discuss what we believe to be the neurophysiologic mechanism behind the slow adducting saccades in internuclear ophthalmoplegia.

Smith and David explained the relatively decreased optokinetic nystagmus amplitude of the affected eye on the basis of their observations in the dysmetria test. When optokinetic targets were moved in the temporal direction of the affected eye, the lateral and medial recti are primarily responsible, respectively, for the slow and fast phases of the nystagmus; the converse is true for the opposite eye. If an abnormally innervated medial rectus produced only a small fast (saccadic) phase while the lateral rectus in the opposite eye produced a relatively normal (larger) fast phase, then the optokinetic nystagmus in the latter eye would have a greater amplitude.

This explanation is not entirely complete. If the only trouble were truncated fast phases in the affected eye, the slow phases would be normal

Fig 1.—Oculogram made with infra-red recording technique showing typical optokinetic amplitude asymmetry in internuclear ophthalmoplegia. Quick phases of abduction are normal in the right eye (OD), but are abnormally small during adducting of the left eye (OS). Note the flattened slow phases in the eye with the reduced amplitude. Rightward (R) and leftward (L) eye movements are as indicated.



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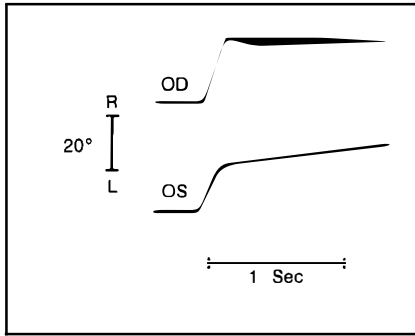


Fig 2.—Simultaneous saccades of abduction (OD) and adduction (OS). Adducting saccade is small and followed by a prolonged tail, whereas the abducting saccade is rapid, overshoots, and then displays a transient nystagmus.

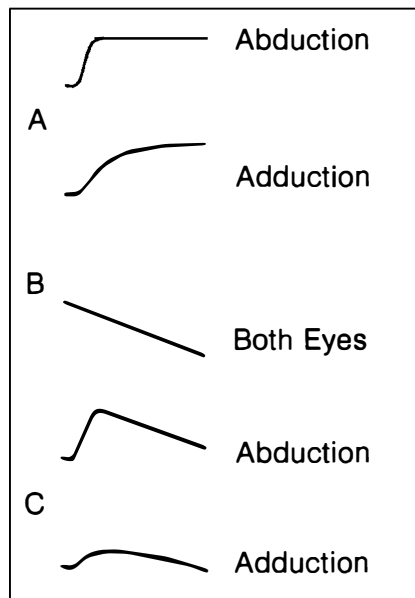


Fig 3.—A diagrammatic explanation of the differential optokinetic response. A, Saccades or quick phases of abduction and adduction; B, smooth following response, which is identical in both eyes; C, two components of nystagmus added together to form a complete cycle. The decreased amplitude in the eye with the adducting quick phase results from the conflict between the tail that follows the small quick phase and the normal attempted following movement.

in size and equal in both eyes, and the result would be an immediate dissociation between the two eyes. The succession of inadequate quick phases in one direction alternating with nor-

mal slow phases in the opposite direction would produce an accumulated change in the position of the affected eye in the direction of the moving targets. This does not occur in these patients. For some reason the slow phases are just as small in size as the abnormally small fast phases (Fig 1). This illustration, which depicts a typical optokinetic nystagmus response in bilateral internuclear ophthalmoplegia was recorded from a patient with multiple sclerosis. The range of adduction was complete in both eyes. The decreased amplitude of both phases of the nystagmus in the eye that adducted during the fast phases is apparent. Also demonstrated is the peculiar flattened appearance of the slow phases.

The explanation for the mechanism of the reduced slow-phase amplitude requires an understanding of the dynamics of saccades in these subtle internuclear ophthalmoplegia cases. Figure 2 shows both abducting and adducting recorded simultaneously. Abducting saccades occur with normal speed and duration. At larger amplitudes they tend to overshoot the target and oscillate transiently. However, with small abducting saccades, such as occur in optokinetic nystagmus, there is no detectable overshoot and they appear normal. Adducting saccades are in sharp contrast. They are characterized by an early rapid portion that carries the eye only a fraction of the way (eg, one third to two thirds) to the target. The remaining distance is covered by a slow movement. It is well established that saccades are created by a pulse-step of innervation.<sup>2</sup> The pulse moves the eye rapidly to the new position; the step holds it there. If the pulse is too small, the quick part of the movement will also be too small. The eye will then continue to move in response to the step but will now move more slowly. The adducting saccade in Fig 2 appears to be created by just such an innervation abnormality. Since the slow adducting movements can sometimes require three seconds before the eye is on target, during which eye velocity is fairly constant, it may comprise both the slow step response and a unocular vergence com-

ponent produced by the disconjugacy present at the end of the pulse of innervation.

Optokinetic nystagmus consists of alternate saccades and slow following eye movements in opposite directions. Since the ocular motor final common path is approximately linear, each cycle can be constructed by the summation of the responses to a step change in target position and the constant target motion in the opposite direction. Figure 3, A and B are graphic reconstructions of each of these two elements of one optokinetic nystagmus cycle for both the abducting and adducting eye of a patient with internuclear ophthalmoplegia. Figure 3C shows how, when added together, they result in the optokinetic nystagmus pattern actually seen differentially between the two eyes in this abnormality. The decreased slow-phase amplitude is created by the competition between the slow terminal portion of the small fast phase and the attempted following movement in the opposite direction. These two components, with approximately equal velocity at the start of the slow phase, result in the flat-topped appearance when net eye velocity is zero. Thus, the net amplitude of the fast and slow phases would be reduced but equal in size, with the frequency of the optokinetic nystagmus being identical to that in the other eye.

This same explanation should apply for the similar unocular amplitude reduction of postrotary nystagmus recently reported in internuclear ophthalmoplegia.<sup>3</sup> Unfortunately, the small time constant employed in the alternating current recording of eye movements in this study obscured the details of the wave shapes of the attenuated nystagmus.

## References

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