Reprint & Copyright © by
Aerospace Medical Association, Washington, DC

Clinical Medicine

Braking Saccade—A New Fast Eye Movement

L. F. DELL'OSSO and R. B. DAROFF

Ocular Motor Neurophysiology Laboratory, Miami Veterans Administration Hospital; and Department of Neurology, University of Miami, School of Medicine, Miami, Florida 33125

DELL'OSSO, L. F., and R. B. DAROFF. *Braking saccade—A new fast eye movement.* Aviat. Space Environ. Med. 47(4):435-437, 1976.

A new type of fast eye movement (FEM) is described whose main function is to stop a runaway slow eye movement (SEM). It conforms to the velocity-amplitude relationship characteristic of other types of saccades. The identification of this FEM is the result of examination of the many waveforms manifested by subjects with congenital nystagmus. It is a common, repeatable phenomenon present in all subjects with any of six different types of nystagmus waveform. The fact that braking saccades reset SEM to zero velocity, whereas no other type of saccade interacts with SEM, indicates a developmental mechanism in such subjects.

WE RECENTLY listed all the fast eye movement (FEM), slow eye movement (SEM), and vergence eye movement (VEM), types which had been described previously in the literature (4). Subsequently, we have identified a new FEM, the braking saccade, from studies of subjects with congenital nystagmus (CN). We have verified that braking saccades have the same velocity-amplitude relationships as the other FEM (2, 10) and differ only in the input responsible for their initiation.

In CN, the eyes move away from a stationary target consequent to an instability in the SEM subsystem (7). At times, the SEM drift off the target is interrupted and the target refoveated by a single large saccade, constituting classical saw-tooth jerk nystagmus. Since the SEM drift is unaltered in saw-tooth nystagmus (congenital, acquired, or induced), the saccadic fast phase is not a "braking saccade." However, a sawtooth waveform is unusual in CN where, more commonly a small amplitude saccade serves only to stop (brake) the eccentric SEM drift and the target is then refoveated by a SEM (Fig. 1). In some CN waveforms, the refoveating SEM actually bypasses the target and a small braking saccade both halts the runaway and refoveates the target (Fig. 2). Although the 25 Hz bandwidth used for these clinical records makes it difficult to identify a braking saccade in each beat of waveforms such as those of Fig. 1 and 2, the simultaneous use of velocity tracings has verified their presence in every instance for all of the 75 patients recorded to date (5). A record of a different type nystagmus, which has rightward braking saccades on the left extreme of eye movement, was made at a bandwidth of 300 Hz in the laboratory of Dr. Robert Steinman. This record, from which Fig. 3 was taken, shows that the braking saccades may also have dynamic overshoots (1). The braking saccades of Fig. 3 are of the foveating type; the subject's eyes are on target in the interval just after the termination of the leftward recovery from the rightward overshoot of the braking saccade. The directions shown are idiosyn-

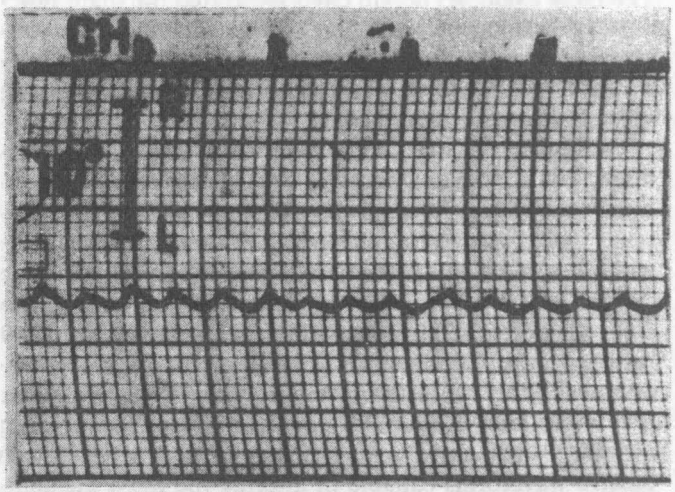

Fig. 1. A form of jerk nystagmus in which leftward braking saccades halt each of the rightward runaways off target. Since these saccades are of insufficient amplitude to refoveate the target, leftward slow eye movements bring the eyes back on target. Target foveation occurs at the rounded left extreme of this waveform. Timing markers are at 1-s intervals.

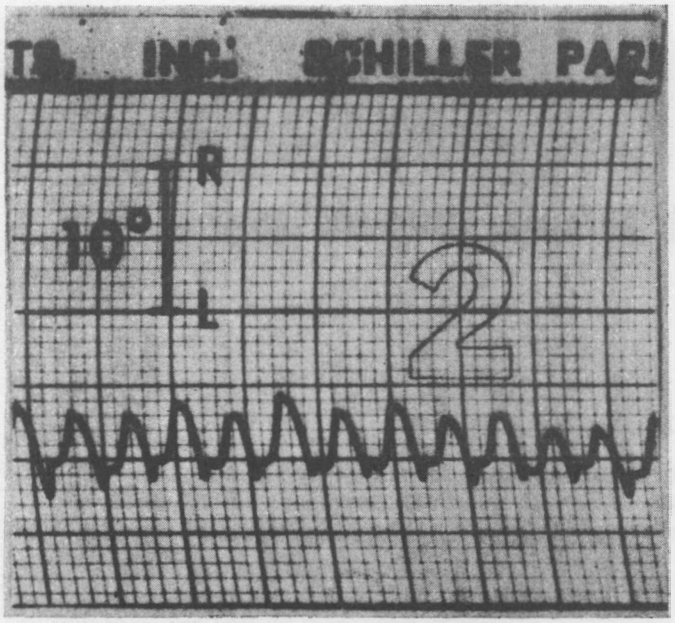

Fig. 2. A form of bidirectional jerk nystagmus in which very small leftward braking saccades halt each of the rightward slow eye movement (SEM) runaways off target and larger, variable-amplitude rightward braking saccades halt the leftward SEM runaways and simultaneously refoveate the target. Target foveation occurs during the motionless segment following the refoveating saccades at the left extreme of the waveform. Time markers are at 1-s intervals.

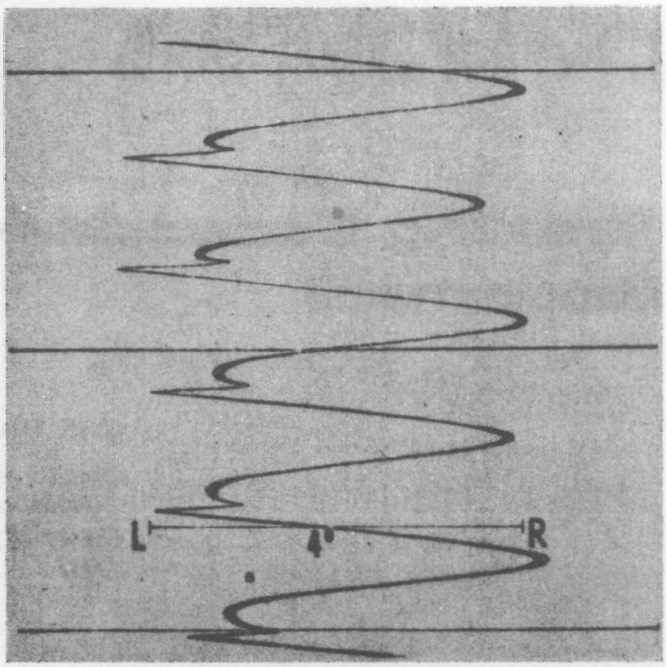

Fig. 3. A form of pendular nystagmus in which the target is foveated by rightward braking saccades which halt the leftward slow eye movement runaway of the eye oscillation. Target foveation occurs at the completion of the leftward return saccade which follows the braking saccade's dynamic overshoot. Time moves upward and time lines are at 1-s intervals.

cratic for this subject; other subjects might exhibit just the opposite. For this type of CN waveform, however, saccades are only present at one extreme of the waveform, not both. Why the SEM in one direction can be halted and reversed without a saccade but those in the opposite direction cannot is not known at present. There are many other CN waveforms which utilize braking saccades and these are discussed in detail elsewhere (5). The three examples herein cited illustrated the main functions of braking saccades; they either simply halt a runaway or simultaneously halt the runaway and refoveate the target. Vision is not a required stimulus as braking saccades also occur in the dark.

Our studies of over 75 patients with CN, using laser-target retinal cinematography and accurate eye movement recordings, have shown that all saccades incorporated in CN waveforms, including braking saccades, are, without exception, corrective in direction (3,5). The critical observation which delineates braking saccades from all other FEM is the FEM/SEM interaction that results in the halting of a SEM along with the repositioning of the eyes consequent to the FEM. No other previously identified FEM have this effect. The braking saccade is presumed to be developmental in that such bizarre waveforms capable of altering the SEM instability occur only in CN and never in acquired forms of nystagmus. More study of this strange interaction between the FEM and SEM subsystems is needed to clarify the mechanism involved.

The way in which a braking saccade is programmed and initiated in a direction opposite to the on-going motion of the eyes is of interest since the information required, given the latencies from the neural pulse in the brainstem to the eye movement, precludes all but an extremely fast proprioceptive loop or the utilization of internal brainstem monitoring (8) of both efferent position and velocity information. Based on the latencies involved in a proprioceptive loop (9), we regard the internal brainstem monitor as the most likely mechanism. By monitoring the efferent position command (i.e., neural integrator output) and comparing it with the desired eye position reference (i.e., derived from either retinal signal or willed eye position) the monitor can program a saccade to correct for any position error. In CN, the monitor should be able to predict the extent of the ocular overshoot consequent to the runaway SEM so that a saccade of the appropriate amplitude will occur at the proper instant to return the eyes to the target. The same process may also explain normal corrective saccades resulting from dysmetric eye movements (11) and the corrective saccade that occurs in macro square wave jerks (8). The reasons for the asymmetry in the control of runaway SEM in patients with CN are obscure at present. Each patient appears to have a preferred bias direction as an inherrent part of his CN waveform; bias reversals do occur in most patients (5).

The identification of the braking saccade demonstrates how careful analysis of a clinical disturbance can provide insights into normal physiological mechanisms.

REFERENCES

1. Bahill, A. T., M. R. Clark, and L. Stark. 1975. Dynamic overshoot in saccadic eye movements is caused by neurological control signal reversals. *Exp. Neurol.* 48:107-122.
2. Boghen, D., B. T. Troost, R. B. Daroff, L. F. Dell'Osso, and J. E. Birkett. 1974. Velocity characteristics of normal human saccades. *Invest. Ophthal.* 13:619-623.
3. Dell'Osso, L. F. 1973. Fixation characteristics in hereditary congenital nystagmus. *Am. J. Optom.* 50:85-90.
4. Dell'Osso, L. F., and R. B. Daroff. 1974. Functional organization of the ocular motor system. *Aerospace Med.* 45:873-875.
5. Dell'Osso, L. F., and R. B. Daroff. 1975. Congenital nystagmus waveforms and foveation strategy. *Docum. Ophthal.* 39:155-182.
6. Dell'Osso, L. F., J. T. Flynn, and R. B. Daroff. 1974. Hereditary congenital nystagmus: An intrafamilial study. *Arch. Ophthal.* 92:366-374.
7. Dell'Osso, L. F., G. Gauthier, G. Liberman, and L. Stark. 1972. Eye movement recordings as a diagnostic tool in a case of congenital nystagmus. *Am. J. Optom.* 49:3-13.
8. Dell'Osso, L. F., B. T. Troost, and R. B. Daroff. 1975. Macro square wave jerks. *Neurology* 25:975-979.
9. Robinson, D. A. 1973. Models of the saccadic eye movement control system. *Kybernetik* 14:71-83.
10. Sharpe, J. H., B. T. Troost, L. F. Dell'Osso, and R. B. Daroff. 1975. Comparative velocities of different types of fast eye movements in man. *Invest. Ophthal.* 14:689-692.
11. Weber, R. B., and R. B. Daroff. 1972. Corrective movements following refixation saccades: Type and control system analysis. *Vision Res.* 12:467-475.