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Saccadic Latency Measurements in Dementia

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• **We measured saccadic latencies in patients with Alzheimer's dementia (AD) and other types of dementia. The saccadic latencies for both groups were considerably longer than those for age-matched controls. The prolongation was as extensive in patients with other types of dementia as it was in those with AD. There was no correlation between latency and the severity of the dementia.**

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Early laboratory examination of patients with progressive intellectual deterioration can sometimes identify a reversible cause of dementia.[1-4] Nevertheless, autopsy studies have shown Alzheimer's dementia (AD) to be the most common cause of dementia in the elderly.[5] Currently, no sensitive or specific diagnostic tool is available to distinguish AD from other types of dementia. Pirozzolo and Hansch recently reported that saccadic latencies (SLs) patients with AD were prolonged when compared with those in age-matched controls.[6] They suggested that the magnitude of the SL increase correlated with the severity of the dementia. However, these authors did not address the question of whether ocular motor reaction time measurements are useful in the differential diagnosis of dementia. To determine whether this noninvasive procedure would be useful as a diagnostic tool, we measured SLS in patients with AD and in those with other kinds of dementia.

PATIENTS AND METHODS

We studied 11 patients whose intellectual impairment satisfied *DSM-III* criteria

Cognitive Test Scores, Functional Impairment, and SLs in Patients With Dementia*

Patient/Age, yr/Sex	Cognitive Test Scores			SL	Diagnosis
	CCSE†	MMSE†	FAQ‡		
1/82/F	15	17	29	399	AD
2/73/F	6	17	30	336	AD
3/84/F	6	19	28	317	AD
4/71/F	9	. . .	13	389	AD
5/60/F	17	25	12	283	AD
6/67/M	14	18	15	394	AD
7/69/F	23	. . .	11	257	AD
Mean age, 72.3	12.9	19.2	19.7	339	. . .
8/59/F	13	13	21	305	Temporal lobe glioma
9/68/M	14	. . .	7	370	Alcoholic dementia
10/77/F	11	. . .	21	396	Multi-infarct dementia
11/73/F	11	16	17	276	Multi-infarct dementia
Mean age, 69.2	12.2	14.5	16.5	337	. . .

*SLs indicates saccadic latencies; CCSE, Cognitive Capacity Screening Examination[11]; MMSE, Mini-Mental State Examination[12]; FAQ, Functional Activities Questionnaire[10]; and AD, Alzheimer's dementia.

†A score of 20 or less is consistent with an organic mental syndrome.

‡A score of 30 represents complete dependency; a score of 0 represents no functional impairment.

for dementia. Clinical histories were obtained not only from patients but from spouses, relatives, and other observers. Particular attention was paid to past or present use of alcohol or other drugs. Complete physical, neurologic, and laboratory examinations were carried out in each case.

Seven patients had had an insidious onset of intellectual and functional impairment with progressive deterioration. Other diseases or conditions known to cause dementia had been ruled out. These patients were assumed to have AD. One woman had occasional deficits in verbal comprehension in addition to intellectual deterioration and was found to have a left temporal lobe glioma. One patient had alcoholic dementia, and two had multi-infarct dementia, according to clinical criteria outlined by Selzer and Sherwin.[8]

Patients ranged in age from 59 to 84 years (mean, 71.2 years). The mean age of the AD group (72.3 ± 8.4 years) did not differ significantly from that of the non-AD group (69.2 ± 7.7 years). The age-matched controls without dementia ranged in age from 59 to 87 years (mean, 72.0 ± 7.9 years). Characteristics of their eye movements have been reported elsewhere.[9]

A research nurse interviewed each patient and at least one other family member, using the Functional Activities Questionnaire (FAQ).[10] Most participants completed two brief cognitive function tests, the Cognitive Capacity Screening Examination (CCSE)[11] and the Mini-Mental State Examination (MMSE).[12]

Eye movements were measured with the infrared reflection technique, which measured both position and velocity. Measurements were recorded on a modified strip chart recorder (Beckman Type R rectilinear Dynograph). The total system bandwidth (position and velocity) was DC to 100 Hz. Patients sat at the center of a 1.5-m-radius arc, with a chin rest and headband for stabilization. Light-emitting diodes were mounted in the arc at angles of 0° and ± 3°, 5°, 10°, 15°. and 20°. These were illuminated in a random sequence by a computer (Digital Equipment Corporation MINC-11). Saccadic latency was defined as the time between illumination of the diode and the instant at which the subject's eye movement angular velocity exceeded 5°/s. A total of 140 stimuli were presented, and saccades were recorded and simultaneously digitized for subsequent off-line analysis. Saccades not related to a stimulus were eliminated from the raw data by a program that discarded saccades spanning less than 3° of arc or with latencies of less than 100 ms or greater than 750 ms. The remaining saccades for each subject were then averaged (range, 46 to 109 saccades per subject). All subjects were given frequent verbal encouragement to help them remain alert and to keep them from blinking excessively. The engineers performing SL measurements were unaware of the patients' clinical diagnoses.

RESULTS

We found prolonged SLs in patients with AD (mean, 339 ± 57 ms) and in those who were demented for other reasons (mean, 337 ± 55 ms). There were no notable differences between these two groups in the prolongation of SLs or in measurements of cognitive or functional impairment (Table). These SLs were much longer than those measured in nondemented subjects of similar age (275 ± 75 ms).[9]

There appeared to be no correlation between SL prolongation and cognitive impairment, as estimated by CCSE scores ($r = .32$) or MMSE scores ($r = .17$). There was no correlation between SL prolongation and functional impairment, as estimated by FAQ scores ($r = .16$). This lack of correlation held true for the subgroups (AD and other dementias) as well as for the demented patients as a whole.

COMMENT

Growing public awareness of the variety of causes of intellectual impairment in the elderly pressures today's physician to arrive at a more specific clinical diagnosis than "dementia" or "organic brain syndrome." As in other progressively debilitating illnesses, families also urge physicians to estimate the severity of the disease process. Pirozzolo and Hansch[6] reported that SLs were prolonged in patients with AD and that they correlated well with the severity of the disease. They even suggested a regulatory role for the cerebral cortex in sensorimotor integration, based on their findings.

We also found that SLs were prolonged in patients with dementia affecting the cerebral cortex. However, our results differed from those of Pirozzolo and Hansch[6] in that we found no correlation between SL prolongation and the severity of cognitive and/or functional impairment. Neither did we find prolonged SLs to be specific for any particular type of dementia. Therefore, we cannot recommend SL measurements as a tool in the diagnostic workup of the demented patient.

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