Article ▸ Oculomotor Dysfunction: Where's the Evidence?

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ABSTRACT

Oculomotor dysfunction (OMD) is a common optometric differential considered among children who demonstrate below-average reading fluency or eye-hand coordination, despite the paucity of high-quality evidence regarding its true etiology, impact on quality of life, and responsiveness to treatment. This viewpoint offers a critical look at the quality of evidence surrounding OMD and questions what we really know about this disorder.

Keywords: learning-related vision problem, literature review, oculomotor dysfunction, oculomotor training

Introduction

Oculomotor dysfunction (OMD) is a deficiency of eye movement ability relative to age or developmental status that consists of smooth pursuits, tracking eye movements used to follow a moving target; and saccades, a series of step-like fixations utilized for scanning and reading. Recognizing that oculomotor control naturally improves with age and becomes more important when reading multiple lines of text, OMD does not typically apply to younger children still learning to read,[1] but rather appears to be most apparent in those with normal intelligence and linguistic ability who struggle with reading fluency.[2] Studies relating to OMD arise from numerous disciplines and are of particular interest in the fields of optometry, reading disability, brain research, and neurorehabilitative science. Because eye movements are controlled by various communicating areas of the brain and are heavily influenced by executive decisions and attentional processes, interpretation of these studies in the context of optometric intervention is challenging. While much of the literature presumes poor eye movements to be a manifestation of more centralized dysfunction (Table 1), few recognize OMD as a distinct pathogenic entity, which adds to the difficulty of locating studies that appropriately address its natural history, diagnosis, and management. The original studies referenced in the American Optometry Association's practice guideline involving the management of OMD[3] suffer similar limitations of relevance (Table 2). As an example, the authors provided a list of symptoms commonly associated with OMD yet cited no direct evidence to support it; similarly, they wrote that "at the conclusion of therapy, ocular motility should be more accurate, and the incidence of accompanying head and body movement [should be] lower (p. 34)," yet they fail to reference original studies that address any form of treatment for OMD. This lack of suitable evidence should encourage clinicians to exercise caution when utilizing diagnostic tools or therapeutic protocols that have not yet withstood rigorous testing.

Etiology

Identifying an underlying cause of dysfunctional eye movements is critical to determining whether the dysfunction requires intervention, which discipline would best manage it, and whether it will respond to treatment. To emphasize the potential influence of underlying pathology, OMD is hereafter divided into primary and secondary dysfunctions. Primary dysfunction can be thought to occur when reading or tracking demands outpace the development of oculomotor control in an otherwise healthy individual. In contrast, secondary OMD results from pathologic dysfunction of or injury to neurologic processes that ultimately compromise the quality of eye movements. Possible etiologies include motor or cognitive delay, linguistic or attention deficit, neurologic disease, traumatic brain injury, and vergence dysfunction. Without considering these factors, the clinician risks prescribing an intervention that may not directly address the root of the dysfunction.

While it appears that primary OMD is frequently encountered in clinical practice, there is a void of literature that describes the origin or natural history of delayed maturation of eye movements in otherwise healthy children. Normative studies of oculomotor function clearly illustrate a correlation between age and oculomotor control in healthy patients,[1,4-5] but controlled trials are needed to clarify the functional impact and course of disease in those diagnosed with primary dysfunction.

Saccadic eye movements interact closely with the vergence system during reading. Several studies[6-9] implicate vergence dysfunction as a viable cause of poor binocular coordination or saccadic dysfunction associated with fluency-related symptoms including loss of place, re-reading, and skipping words or lines of text.[3] This notion is supported by findings of improved binocular coordination and oculomotor performance after resolution of the vergence deficit.[9-11] Gaertner et al.[9] found in a small sample of children who demonstrated poor binocular saccadic coordination that orthoptic enhancement of their fusional vergence amplitudes also improved their oculomotor control, emphasizing the influence of vergence function on saccadic control. Gallaway and Boas[10] demonstrated similar findings of improved oculomotor control in patients with vergence dysfunction following completion of an orthoptic regimen that excluded oculomotor-specific training. Ramsay et al.,[11] working under the observation that dyslexic individuals tend

Table 1. Compilation of Original Studies Investigating the Etiology of Poor Oculomotor Control

Study Title	Publication Date	Mechanism Implicated
Saccades of hyperactive and normal boys during ocular pursuit[18]	1981	Poor attention control
Eye movements, prematurity, and developmental co-ordination disorder[13]	1998	General motor deficit
Role of visual attention in cognitive control of oculomotor readiness in students with reading disabilities[20]	2001	Poor attention control
Altered control of visual fixation and saccadic eye movements in attention-deficit hyperactivity disorder[19]	2003	Difficulty inhibiting saccadic intrusions
Saccade control in dyslexia: development, deficits, training and transfer to reading[21]	2008	Deficit in anti-saccade control
Dyslexic children are confronted with unstable binocular fixation while reading[36]	2011	Deficit in vergence-saccade interaction
Immaturity of the oculomotor saccade and vergence interaction in dyslexic children: evidence from a reading and visual search study[22]	2012	Deficit in vergence-saccade interaction
Smooth pursuit eye movements in children with strabismus and in children with vergence deficits[5]	2013	Vergence deficit
Analysis of saccades and peak velocity to symmetrical convergence stimuli: binocularly normal controls compared to convergence insufficiency patients[6]	2013	Vergence deficit leading to saccadic disconjugacy
Binocular coordination of saccades during reading in children with clinically assessed poor vergence capabilities[7]	2013	Vergence deficit leading to saccadic disconjugacy
Association between reading speed, cycloplegic refractive error, and oculomotor function in reading disabled children versus controls[8]	2013	Deficit in vergence facility
Vertical and horizontal smooth pursuit eye movements in children with developmental coordination disorder[12]	2014	Delayed maturation of smooth pursuits
Vertical saccades in dyslexic children[24]	2014	Impairment in cortical areas responsible for vertical saccades
Binocular saccade coordination in reading and visual serach: a developmental study in typical reader and dyslexic children[25]	2014	Poor attention processing and deficit in vergence-saccade interaction
Eye movements of university students with and without reading difficulties during naming speed tasks[27]	2014	Impaired reading ability
The influence of oculomotor tasks on postural control in dyslexic children[28]	2014	Poor attention control
Evaluation of ocular movements in patients with dyslexia[29]	2015	Deficit in visual processing of linguistic material
Immaturity of visual fixations in dyslexic children[23]	2016	Poor attention control and/or impairment in cortical areas responsible for fixation

to have subnormal saccadic control, found in a sample of dyslexic children that a 5-week computerized vergence training program improved reading speed.

OMD has received particular interest from investigators looking for readily apparent indicators of generalized motor delay, formally termed developmental coordination disorder (DCD).[12-13] Performance-related deficits demonstrated in children with DCD include those often attributed to smooth pursuit dysfunction, such as difficulty hitting or catching a ball.[3] Langaas et al.,[14] in addition to finding abnormal pursuit eye movements in patients with DCD, demonstrated that they struggled to synchronize their motor output appropriately with visual stimuli. They concluded that dysfunctional eye movements in the context of developmental motor difficulty are more likely an extension of corporal deficiency in predictive motor control rather than the primary cause of poor eye-hand coordination.

Evidence suggests that visual attention is closely tied to eye movements.[15-17] Shepherd et al.[17] concluded that a shift in attention from one target to another is prerequisite to a corresponding shift in fixation; in the same way, maintaining fixation on a stationary or moving target requires continuous visual attention to that target. Patients with poor attentional control have difficulty attending exclusively to a given task or target for any extended length of time, and their quality of eye movements, whether in the form of inefficient saccadic behavior or interrupted smooth pursuits, may reflect this tendency.[18-19] Solan et al.[20] reported in a study of reading-disabled adolescents that oculomotor training and reading remediation equally and independently improved measures of both saccadic efficiency and reading comprehension. They suggested that improved visual attention was the prevailing mechanism common to both interventions, as well as generalized effects of receiving therapy, such as increased intrinsic motivation and access to individual support.

There is an abundance of research and controversy surrounding eye movements in patients with reading disability, particularly dyslexia. Depending on the perspective of the authors, two opposing assumptions appear in the literature: first, that poor oculomotor control associated with dyslexia additionally impacts reading performance; and second, that apparent oculomotor dysfunction is simply a reflection of impaired linguistic processing and does not independently contribute to reading difficulty. The idea that dyslexia-associated

Table 2. Investigative Studies Related to Oculomotor Dysfunction Referenced in the American Optometric Association's Clinical Guideline for Learning-related Vision Problems[3]

Study Title	Publication Date	Sample Size	Sample Profile	Limitation of Results*
Eye movements during reading: case reports**	1976	5	Adults with neurologic disease	CG,RI,SS
Saccadic intrusions contributing to reading disability: a case report	1983	1	Adult with neurologic disease	CG,RI,SS
NYSOA K-D test	1983	1202	Normal subjects	NC,RI
Eye movements during reading: further case reports	1985	4	Adults with neurologic disease	CG,RI,SS
Interrater and test-retest reliability of pursuits and saccades	1988	***	Normal subjects	NC
A new visual-verbal saccade test: the developmental eye movement test (DEM)	1990	***	Normal subjects	NC
Saccadic eye movements of dyslexic and normal reading children	1994	24	Dyslexic children	SS
Poor saccadic control correlates with dyslexia	1998	185	Dyslexic subjects 8-25 years	
Midline-crossing inhibition: an indicator of developmental delay	1999	34	Subjects with developmental delay	SS,UR
Voluntary saccadic control in dyslexia	2000	620	Dyslexic children 7-17 years	
Stability of gaze control in dyslexia	2000	361	Dyslexic children 7-17 years	

*Limitations Key:
CG: no control group
NC: subjects were taken from a normal cohort
RI: study is over 30 years old and risks irrelevance against more recent publications
SS: small sample size (< 50 subjects)
UR: study is not directly related to the visual system

**Reference numbers match those listed in the clinical guideline referenced
***Sample size not listed in abstract

OMD exacerbates reading difficulty is supported by evidence suggesting that abnormal or immature oculomotor pathways are common in patients with dyslexia and may impair eye movement function.[21-25] Evidence also supports the inverse relationship, in which impaired familiarity with sight words and delayed phonetic processing significantly impact planning and execution of saccades.[26-30] While these competing theories can potentially coexist, further investigation with controlled trials is needed to determine whether vision training, reading remediation, or a combination of therapies would most appropriately address OMD in the context of reading disability. Determining the most effective treatment may in turn reveal the predominant mechanism of OMD in this cohort.

Diagnosis

Historically, diagnosis of OMD relied on qualitative observation of pursuits and saccades, or on results of the Developmental Eye Movement (DEM) test. While large-scale studies have obtained normative values for these methods,[1,31] no similarly-sized efforts have attempted to link symptoms attributed to OMD with those who fall outside the norms. In fact, recent research has sufficiently discredited the DEM test as having poor reliability and little association with saccadic eye movements or reading performance.[32-35] Qualitative observation has not been afforded such rigorous testing of validity, and its correlation with symptoms and reading performance remains unverified.

With the advent of infrared eye-tracking technology, it is possible to precisely quantify the various components of eye movements used during reading. Numerous studies demonstrate that certain components of saccadic eye movements correlate with aspects of reading performance.[36-38] However, correlation alone does not clarify the direction of causality between poor eye movements and reading difficulty, and interpretation of these studies should acknowledge this ambiguity. Tannen and Ciuffreda[38] aimed in a small case series to differentiate a linguistic from a visual origin of OMD by having subjects read paragraphs both at their current reading level and several levels below. They suggested that a dramatic improvement in saccadic performance with easier text indicated a linguistic etiology, whereas little to no improvement implied a true oculomotor dysfunction. Larger-scale controlled studies are needed to validate this method.

Treatment

Conventional management of OMD involves repetition of visual tasks that require increasing levels of accuracy and speed of eye movements. Treatment of pursuit dysfunction involves continuous-motion eye-tracking activities, often with integration of whole-body motor tasks, whereas saccadic training incorporates more precise scanning, target localization, and reading tasks.[2] In the absence of an evidence-based treatment protocol, therapeutic programs vary widely in content, structure, and duration. While several studies have shown that pursuits and saccades can improve with oculomotor training,[39-46] no large-scale, randomized controlled trials have demonstrated its efficacy for clinically-documented OMD. In fact, studies investigating oculomotor training for primary OMD are conspicuously absent from the literature, which highlights a concerning disconnect with the apparent

Table 3. Compilation of Original Studies Investigating the Efficacy of Oculomotor Training in Improving the Quality of Eye Movements

Study Title	Publication Date	Sample Size	Sample Profile	Results	Limitation of Results*
The effect of oculomotor training on reading efficiency[39]	1991	10	College students with reading difficulty	Improved oculomotor control on Visagraph	SS,Cr
Effects of visual training on saccade control in dyslexia[40]	2000	85	Dyslexic subjects 8-15 years old	33% improved reading scores by 1-2 percentile classes	II
Role of visual attention in cognitive control of oculomotor readiness in students with reading disabilities[20]	2001	31	6th grade students with reading disability	Oculomotor training and reading therapy had similar improvement in oculomotor control	SS
Reading-related oculomotor testing and training protocols for acquired brain injury in humans[41]	2004	14	Adults with acquired brain injury	Reading rate improved by average of 25%	SS
Saccade control in dyslexia: development, deficits, training and transfer to reading[21]	2008	21**	Dyslexic subjects 7-17 years old	Reading errors decreased by 45% vs. 20% in dyslexic controls	SS
Sustained effects for training of smooth pursuit plasticity[42]	2012	20	Normal adults	Improved quality of smooth pursuits	Cr,NC,SS
Versional eye tracking in mild traumatic brain injury (mTBI): effects of oculomotor training (OMT)[43]	2014	12	Adults with mTBI	Significant reduction in saccadic inaccuracy compared to controls	Cr,CG,SS
The effect of saccadic training on early reading fluency[44]	2014	96	Normal subjects 5-8 years old	Improvement of 17 percentile points on reading fluency score in treatment group vs. controls	Ce,CI,NC
The effect of in-school saccadic training on early reading fluency[45]	2016	327	1st and 2nd grade students (148 normal subjects and 79 on IEP)	Reading fluency and comprehension scores improved by 6% and 8% vs. 4% and 2% in controls	Ce
Vision therapy for post-concussion vision disorders[46]	2017	18***	Subjects 6-72 years with history of concussion	83% improved DEM scores	Cr,CG,SS

*Limitations Key:
Ce: change in eye movements not measured in study outcomes
Cr: change in reading efficiency or other performance-based metric not measured in study outcomes
CG: no control group
CI: primary author disclosed significant conflict of interest in study
II: intervention was ineffective in majority of subjects
NC: sample was taken from a normal cohort
SS: small sample size (< 50 subjects)
**Sample was taken from a larger cohort of 3,344 subjects, of which 182 underwent vision training, of which 21 underwent reading testing after training
***Sample was taken from a larger cohort of 218 subjects, of which 18 were treated for saccadic dysfunction

casualness with which primary OMD is diagnosed and treated in clinical practice. Furthermore, a majority of the available studies on secondary OMD provide sufficient reason for caution in their interpretation or application of findings (Table 3), including in some the absence of any attempt to correlate their results with a change in reading performance or symptoms. Despite study limitations, the gains in oculomotor control among specific cohorts should not be categorically dismissed. Regarding subjects with a history of traumatic brain injury, Han et al.[41] found a 25% average improvement in reading pace, and Thiagarajan and Ciuffreda[43] demonstrated improved saccadic accuracy following oculomotor training. Gallaway et al.[46] found in a similar cohort that saccadic dysfunction resolved in 83% of affected patients, although the now-controversial DEM test was the only tool used for diagnosis and assessment of treatment effect, and vergence training was included in the therapeutic program. In patients with dyslexia, Fischer and Hartnegg[40] found that one third of their cohort improved standardized reading scores by 1-2 percentile classes after oculomotor training; in a separate study, they demonstrated a significant reduction of reading errors compared to dyslexic controls.[21] Solan et al.[20] noted similar improvement in reading performance following oculomotor training compared with reading remediation; however, as discussed above, the mechanism of improvement for these patients remains unclear. While these findings are encouraging, better research is needed to support the notion that attempting to improve oculomotor control in patients with OMD results in a meaningful improvement in reading fluency or performance.

Conclusions

The diversity of etiological possibilities behind inefficient eye movements challenges conventional optometric belief that any oculomotor dysfunction can be remediated with oculomotor therapy. Even in instances of apparent primary dysfunction, the available literature does not provide definitive proof of functionally meaningful treatment effect. As for secondary dysfunction, there is little reliable evidence to justify the prescription of oculomotor therapy as a non-inferior alternative or viable supplement to interventions that

may more directly or comprehensively address the associated underlying disorder. While the sharing of clinical wisdom and small-scale experimentation are essential to the development of paradigm-changing practice patterns, the optometric community must acknowledge the deficiency of relevant, high-quality evidence regarding OMD and, aspiring to the successes of the Convergence Insufficiency Treatment Trials, collectively advance toward scientifically validating that which we presume to know about this disorder.

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