CONDITION

Cogan type ocular motor apraxia

Learn how Cogan type oculomotor apraxia affects voluntary eye movements, why head thrusts occur, how it is diagnosed, and what support may help.

WHAT IT IS
Rare eye movement disorder impairing voluntary saccades

BODY SYSTEM
Nervous system

MAIN SYMPTOMS
Delayed eye movements, rapid head turns, blinking before sideward gaze, difficulty following fast objects

WHAT CAUSES IT
Unknown, sometimes genetic

HOW IT IS DIAGNOSED
Eye movement examination, exclusion of other conditions

MAIN TREATMENT
Supportive therapies and visual accommodations

CAN IT BE CURED?
No, no established treatment restores saccades

IS IT AN EMERGENCY?
Sometimes, sudden eye movement difficulty with one-sided weakness

LONG-TERM OUTLOOK
Generally nonprogressive, though underlying disorders may progress

Overview

Cogan type oculomotor apraxia is a rare eye movement disorder that usually begins in infancy or early childhood. It is also called congenital ocular motor apraxia, or COMA.

A person with this condition has difficulty starting fast, purposeful eye movements called saccades . Saccades allow the eyes to shift quickly from one object to another, such as from a book to a person across the room. Horizontal movements from side to side are usually affected more than vertical movements.

The eye muscles are not necessarily weak or paralyzed. Instead, the nervous system has trouble starting the intended movement. The word "apraxia" generally means difficulty carrying out a learned movement, although Cogan type oculomotor apraxia is more precisely

considered a problem with starting saccades.

Children often compensate by blinking or making quick head turns, sometimes called head thrusts. These movements help bring the object of interest into view. They may become noticeable only after an infant develops enough head control to look toward people and objects.

Cogan type oculomotor apraxia may occur by itself. However, similar eye movement findings can occur with genetic, metabolic, or neurological conditions. A careful evaluation is therefore important, especially when a child also has poor coordination, unusual muscle tone, developmental differences, or loss of previously acquired skills.

This condition is not the same as Cogan syndrome , an inflammatory disorder involving the eyes and inner ears. It is also different from the progressive disorders called ataxia with oculomotor apraxia.

Symptoms

The most recognizable sign is difficulty looking quickly toward a new target. When asked to look to one side, a child may initially keep the eyes near the center rather than moving them directly toward the object.

To compensate, the child may turn the head rapidly toward or beyond the target. The normal reflex linking head and eye movement helps reposition the eyes. Once the target is located, the head may return to a comfortable position. Blinking may also help start the eye movement.

Possible features include:

The pattern varies. Some children have mainly an eye movement problem and otherwise typical development. Others have additional neurological findings that may point to a broader condition.

Visual acuity-the ability to see detail-may be normal. However, difficulty directing the eyes can affect how efficiently a child uses vision. Reading, copying from a board, navigating busy spaces, and some sports may require extra time or head movement.

The compensatory head movements often become less dramatic with age. This may happen because saccade control improves or because the child develops subtler strategies. Improvement does not always mean the underlying eye movement difference has completely resolved.

- Repeated, rapid head turns when shifting attention
- Blinking before attempting to look to the side

- Delayed or incomplete side-to-side eye movements
- Difficulty following fast-moving objects
- Trouble moving the eyes between words, lines, or visual targets
- An unusual head position during visual tasks
- Clumsiness, delayed walking, or balance difficulty in some children
- Low muscle tone, developmental delay, or speech and language delay in some cases

Causes and risk factors

The exact cause of isolated Cogan type oculomotor apraxia is often unknown. It appears to involve brain networks that plan and start rapid eye movements. These networks include areas of the brainstem and cerebellum, the part of the brain that helps coordinate movement and balance.

Some families have more than one affected person, suggesting that genetic factors can sometimes contribute. Different inheritance patterns have been reported, but there is no single gene or inheritance pattern that explains every case labeled Cogan type oculomotor apraxia.

There are no established lifestyle causes. Parents should not assume that ordinary activities, diet, or routine decisions during pregnancy caused the condition.

An important part of evaluation is determining whether the eye movement pattern is isolated or belongs to another disorder. Similar findings may occur with conditions such as Joubert syndrome, certain cerebellar disorders, ataxia-telangiectasia, and the inherited ataxia with oculomotor apraxia syndromes. Some of these conditions are progressive, while classic isolated Cogan type oculomotor apraxia is generally not considered a progressive neurodegenerative disease.

Factors that may increase the likelihood of an underlying genetic or neurological condition include:

These findings do not identify a specific diagnosis on their own. They help clinicians decide what further testing may be useful.

- A family history of similar eye movements, unexplained poor coordination, or early neurological disease
- Developmental delay affecting several areas
- Loss of skills that had already been learned

- Progressive balance or coordination problems
- Abnormal muscle tone, weakness, seizures, or unusual breathing patterns
- Additional abnormalities found on a neurological examination or brain scan

Diagnosis

Diagnosis begins with a detailed history and examination. Parents may be asked when the head movements first appeared, whether they are changing, and whether development has followed the expected course. Home videos can be useful because the movements may not occur consistently during an appointment.

A pediatric ophthalmologist or neuro-ophthalmologist may examine:

Preserved reflex eye movements can help show that the eye muscles and basic movement pathways are functioning, even though voluntary saccades are difficult to start. Formal eye movement recording may be used in specialized centers but is not required in every case.

A neurological and developmental assessment can look for coordination problems, abnormal muscle tone, speech delay, learning differences, or other clues. Clinicians may recommend a hearing assessment because hearing difficulty can affect development and responses to targets.

Brain magnetic resonance imaging, or MRI, may be considered. MRI can look for structural differences in the cerebellum, brainstem, or other areas. Imaging may be normal in isolated Cogan type oculomotor apraxia. A normal scan does not rule out the condition.

Genetic testing may be appropriate when there are additional neurological features, an affected relative, an unusual MRI result, or uncertainty about the diagnosis. Testing might use a panel of genes, chromosome analysis, or broader sequencing. A genetics professional can explain what a result may and may not show.

No single test confirms every case. Diagnosis often depends on the characteristic movement pattern and on excluding conditions that require different monitoring or treatment.

- How the eyes move toward a visual or sound target
- Whether horizontal and vertical movements are affected differently
- The child's use of blinking and head thrusts
- Smooth tracking of a slowly moving object
- Reflex eye movements produced by gentle head movement

- Visual acuity, focusing, eye alignment, and the health of the eyes

Treatment and management

There is no established medicine that reliably restores normal saccade initiation in isolated Cogan type oculomotor apraxia. Management focuses on helping the person use vision effectively, addressing related needs, and monitoring for signs of a broader condition.

Compensatory head movements are useful adaptations. A child generally should not be repeatedly told to stop them unless the movements are causing pain or another specific problem. Forcing the head to remain still may make it harder to find visual targets.

An ophthalmologist can identify and treat other eye problems, such as nearsightedness, farsightedness, astigmatism, or strabismus. Strabismus means that the eyes do not point in the same direction. Glasses or other eye care may improve usable vision, but they do not directly correct the saccade initiation problem.

Physical and occupational therapy may help with balance, coordination, safe movement, and daily visual tasks. A therapist may teach strategies such as turning the head in a controlled way, using larger visual targets, reducing visual clutter, and allowing more time to scan the environment.

Speech-language therapy and developmental services may help children who have communication or broader developmental delays. School support can be based on the child's functional needs rather than the diagnosis alone. Helpful adjustments may include extra time, clear spacing between lines of text, reduced copying from distant displays, electronic materials, and seating that makes important visual targets easier to locate.

Eye exercises or "vision therapy" should not be assumed to cure the neurological problem. A qualified specialist may recommend task-specific training, but families should ask what evidence supports a proposed treatment and what realistic goals to expect.

Surgery is not a routine treatment for the saccade problem itself. It may occasionally be considered for a separate condition, such as significant strabismus, after specialist assessment.

Complications

Cogan type oculomotor apraxia does not usually cause blindness by itself. Its main effects come from inefficient visual scanning and the need to compensate with head movement.

Possible difficulties include slower reading, losing one's place on a page, trouble following fast activity, and reduced confidence in crowded or unfamiliar environments. Balance or developmental problems can increase the risk of falls.

Frequent or forceful head turns may sometimes cause fatigue or neck discomfort. A therapist can assess posture and suggest safer movement strategies without removing a compensation the person needs.

Academic or social difficulties can develop if head movements are misunderstood as behavioral or if the child is not given enough time to direct the eyes. Explaining the condition to teachers and caregivers can reduce misunderstanding.

When oculomotor apraxia is part of another neurological or genetic disorder, complications depend mainly on that underlying condition. New loss of skills, increasing unsteadiness, weakness, or other progressive symptoms are not automatically explained by isolated Cogan type oculomotor apraxia and should be evaluated.

Follow up

Routine follow-up may involve pediatric ophthalmology or neuro-ophthalmology, with neurology, genetics, developmental pediatrics, or rehabilitation services added when needed. The schedule depends on age, symptoms, test results, and whether other conditions are being considered.

At planned visits, clinicians may review eye alignment, vision, head movements, balance, school performance, development, and neck comfort. Reassessment is also useful when a child starts reading, changes schools, or begins activities with greater visual demands.

Contact the treating clinician promptly if there is clear worsening, loss of developmental skills, new weakness, increasing falls, seizures, swallowing difficulty, or a new decline in vision.

Sudden difficulty moving the eyes is not the usual presentation of congenital Cogan type oculomotor apraxia. Seek emergency care for a sudden eye movement problem accompanied by facial drooping, one-sided weakness, difficulty speaking, severe headache, confusion, loss of consciousness, seizure, or inability to walk. These symptoms may indicate an acute neurological problem.

Sources



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