



## Oculomotor Apraxia

### **What is oculomotor apraxia?**

Ocular motor apraxia (OMA) is the absence of, or a defect in, the control of voluntary, purposeful eye movement. Children with this condition have difficulty moving their eyes in a desired direction. In other words, their saccades (the quick, simultaneous movement of both eyes in the same direction) are abnormal. Because of this, most patients with OMA have to turn their head quickly in order to start following objects in side gaze. They often thrust their head to compensate for this inability to quickly move their eyes to either side. Typically, vertical eye movements are unaffected.

### **Where is the problem with this condition, in the eyes or in the brain?**

The source of OMA is in the brain. The process of initiating eye movements is a complicated neural pathway involving many different structures. Imaging of the brain with magnetic resonance imaging (MRI) is commonly performed when evaluating OMA. Findings may be normal or may reveal poor development of regions of the brain, in particular: the corpus callosum, cerebellum, and/or fourth ventricle.

### **What other developmental problems can coexist with OMA?**

Extensive investigations sometimes reveal no associated developmental issues. However, children with OMA often have developmental delays and can possess low muscle tone (hypotonia). Speech, reading, and motor delays are common even when imaging studies of the brain are normal.

### **What causes OMA to develop?**

The etiology of OMA is usually not known. However, the condition is sometimes attributed to neurologic problems that occur during early infancy up to the first 6 months of life. Such issues may include perinatal hypoxia, meningitis, periventricular leukomalacia, cerebral palsy, septicemia, anemia, herpes encephalitis, and seizure disorder.

### **What other clinical associations can exist with OMA?**

Sometimes OMA can be seen in patients with underlying conditions. A wide range of clinical entities have been reported in children with OMA. These include agenesis of the corpus callosum, Joubert syndrome, Tay-Sachs disease, Dandy-Walker malformation, microcephaly, hydrocephalus, vermiform hypoplasia, porencephalic cyst, megalencephaly, Krabbe leukodystrophy, Pelizaeus-Merzbacher disease, infantile Gaucher disease, GM1 gangliosidosis, infantile Refsum



disease, abetalipoproteinemia (vitamin E deficiency), Niemann-Pick type C disease, propionic acidemia., ataxia-telangiectasia, Bardet-Biedl syndrome, vermian astrocytoma, vermian cyst, carotid fibromuscular hypoplasia, Cornelia de Lange syndrome, Wilson disease, and microphthalmia.

### **If a child has OMA, do siblings or future children have a risk for the condition?**

The genetics of OMA are not well understood and may be multifactorial. Isolated OMA is generally considered non-hereditary and would not be associated with an increased risk of siblings or other family members developing this condition. However, a number of genetic mutations have been identified which cause OMA in addition to other clinical features. Siblings would be at increased risk of developing the condition in these cases, but the inheritance patterns can be variable.

### **What is the treatment for OMA?**

There is no specific treatment for OMA. However, if a child has OMA, parents should be aware of potential developmental delays that can be associated with this condition., Thus, therapies may be started sooner rather than later when necessary. Serial ophthalmologic examinations are recommended to monitor for other eye problems that can be associated with OMA.

### **Does OMA improve over time and resolve?**

There are few reports of the long-term prognosis of children born with OMA. The head thrusts associated with OMA typically diminish over time but tend not to completely disappear. This may represent a true improvement in the condition, or it might be just be an adaptation over time that masks the head thrust.

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