Isolated fourth nerve palsy
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Introduction

This article includes discussion of isolated fourth nerve palsy, superior oblique palsy, and trochlear nerve palsy. The foregoing terms may include synonyms, similar disorders, variations in usage, and abbreviations.

Overview

Isolated fourth nerve palsy is usually benign. The most common etiologies are congenital, traumatic, ischemic, and idiopathic causes. Extensive evaluations for isolated fourth nerve palsies are generally not indicated, but more intensive evaluation for underlying etiology may be necessary for nonisolated or bilateral fourth nerve palsies. Evaluation of ocular torsion enables differentiation of fourth nerve palsy from skew deviation and other causes of vertical diplopia. Spectacle prisms and strabismus surgery are effective treatments for most unresolved cases.

Key points

- Isolated fourth nerve palsy is usually benign and typically does not require an extensive evaluation.
- Nonisolated fourth nerve palsy should undergo imaging and evaluation directed to the topographically localizing symptoms and signs.
- The most common etiologies for a fourth nerve palsy are congenital, traumatic, ischemic, and idiopathic causes.
- Thyroid-related orbitopathy, skew deviation, and myasthenia gravis can mimic fourth nerve palsy.
- Spectacle prisms and strabismus surgery can be effective treatments for most unresolved cases.

Historical note and terminology

The terms “superior oblique paralysis,” “trochlear nerve palsy,” and “fourth nerve palsy” are essentially interchangeable for the purposes of this review.

Clinical manifestations

Presentation and course

Patients with fourth nerve palsies complain of binocular vertical diplopia and tilting of objects (torsional diplopia). On examination, patients demonstrate an incomitant ipsilateral hypertropia with characteristic features elicited with the 3-step maneuver (Bixenman 1981; Brazis et al 1990; Burde et al 1992; Tamhankar et al 2011a). The first step is to determine the hypertropic (higher) eye. The second step is to determine if the hypertropia is worse in right or left gaze. The third step is to determine if the hypertropia is worse in right or left head tilt. In fourth nerve palsies, the ipsilateral eye is hypertropic (the ipsilateral superior oblique is a depressor; thus, a paresis of this muscle results in an ipsilateral hypertropia). The hypertropia is worse in contralateral gaze, with the affected eye in adduction where the downgaze function of the superior oblique muscle is greatest. The hyperdeviation is worse in ipsilateral head tilt, which stimulates the intorting extraocular muscles of the affected eye (the superior oblique and the superior rectus). The intact superior rectus muscle overpowers the paretic superior oblique resulting in an increase in the hyperdeviation. Therefore, most patients have a compensatory head tilt away from the side of the fourth nerve palsy in order to reduce the ocular misalignment (Kushner 2009). However, some patients have a paradoxical head tilt towards the side of the fourth nerve palsy, presumably to increase the size of the vertical misalignment and allow the patient to ignore the second image more easily (Khan 2005). Many patients do not manifest all of the components of the 3-step test (Manchandia and Demer 2014). Manchandia and colleagues prospectively studied 50 patients with superior oblique palsy, and only 35 (70%) patients fulfilled the entire 3-step test. In 14 (28%) patients, only 2 steps were fulfilled. Among the patients that fulfilled 2 steps, all combinations of the 3 steps were observed.
Ocular movements often appear normal. However, superior oblique palsy can be associated with reduced infraduction in adduction, overaction of the ipsilateral antagonist inferior oblique muscle, and overaction or contracture of the ipsilateral superior rectus muscle (Molinari and Ugrin 2009). Forced duction testing might add additional information for surgical planning in selected cases (eg, absence or laxity of the superior oblique tendon) (Mims 2003). If the patient fixates with the paretic eye (as often happens if it is the better-seeing eye), the normal eye will appear inferiorly displaced.

The superior oblique muscle intorts the eye; therefore, an eye with a fourth nerve palsy is extorted. Extorsion denotes temporal rotation of the 12:00 meridian of the eye. Anatomic extorsion can be assessed by comparing the position of the fovea and the optic nerve on a fundus photograph or during indirect ophthalmoscopy (Phillips and Hunter 1999). In normal patients, a horizontal line through the fovea intersects the inferior half of the optic disc (Bixenman and von Noorden 1982). If the horizontal line intersects below the inferior disc border, the eye is extorted. Conversely, if the horizontal line intersects the superior half of the disc, the eye is intorted. The patient's perception of torsion is assessed by double Maddox rod or Lancaster Red-Green tests.

Differentiating patients with decompensation of congenital fourth nerve palsy from patients with acquired fourth nerve palsy is important. In patients with congenital fourth nerve palsies, the following features may be present: (1) old photos may show a longstanding head tilt; (2) objective fundus excyclotorsion may be seen, but the patients often do not complain of cyclotropia (double Maddox rod torsion testing is normal); (3) vertical fusional amplitudes are greater than 6 to 7 prism-diopters; and (4) there may be facial hypoplasia on the affected side.

Bilateral fourth nerve palsies are suggested by the following features: (1) right hypertropia in left gaze and left hypertropia in right gaze; (2) positive head tilt test to either shoulder (double Bielschowsky test); (3) greater than 10 degrees excyclotorsion on double Maddox rod test; (4) esotropia that is greater in downgaze than in upgaze.

Prognosis and complications

The prognosis for recovery depends on the etiology. Most ischemic palsies recover spontaneously. Other etiologies may not recover spontaneously but are often amenable to correction with spectacle prisms or strabismus surgery.

Clinical vignette

A 60-year-old man with hypertension and diabetes presented with acute, painless, binocular vertical diplopia that was worse in downgaze. The patient also reported tilting of one of the diplopic images (torsional diplopia). On examination, there was a right hypertropia of 4 prism diopters in primary position increasing to 8 prism diopters in left gaze and in right head tilt. There were 5 degrees of excyclotorsion on double Maddox rod testing. The patient displayed head tilt to the left. Duction testing revealed mildly reduced infraduction-in-adduction of the right eye. The other aspects of the neuro-ophthalmic examination were normal. The diagnosis was isolated right fourth nerve palsy of presumed vasculopathic cause. No diagnostic studies were performed. The patient experienced complete resolution of signs and symptoms within 4 weeks.

Biological basis

Etiology and pathogenesis

In children, the most frequent etiologies of isolated fourth nerve palsy are congenital (40%), traumatic (34%), and idiopathic (23%) (Harley 1980; Holmes et al 1999; Mollan et al 2009; Hata et al 2013). Child abuse should be considered in the differential diagnosis of childhood traumatic fourth nerve palsy (Cackett et al 2004).

In adults, decompensated congenital fourth nerve palsy, trauma, and ischemia of the extra-axial fourth nerve predominate. Tumors cause less than 1% of cases (Rush and Younge 1981; von Noorden et al 1986; Mollan et al 2009; Khaier et al 2012; Hata et al 2013).

Bilateral fourth nerve palsies are commonly due to trauma. Rarely, metastatic or other compressive lesions may occur in the dorsal midbrain (Mielke et al 2001).

Bilateral fourth nerve palsy may be due to spontaneous intracranial hypotension (Brady-McCreery et al 2002), and fourth nerve and sixth nerve palsy may occur together after lumbar puncture (Follens et al 2001). It is important to
identify bilateral fourth nerve palsies because surgical repair of only one side will be ineffective in resolving the ocular misalignment, as it brings out the manifestations of the other fourth nerve palsy (Esmail and Flanders 2003).

The etiologies of non-isolated fourth nerve palsy may be differentiated based on clinical topographical localization to the midbrain, subarachnoid space, cavernous sinus, superior orbital fissure, or orbit (Khawam et al 1967; Burger et al 1970; Berlit 1991; Keane 1993; Pollock et al 2002; Veshechek and Spektor 2002; Moulignier et al 2003; Jones et al 2004; Mims 2004; Pinches et al 2004; Sokwala et al 2004; Makki and Newman 2005; Dhalwal et al 2006; Akagi et al 2008; Madigan and Zein 2008). Rare cases of periodic (“cyclic”) fourth nerve palsy have also been reported (Prieto-Diaz and Gallo 2005).

### Table 1. Etiologies for a Fourth Cranial Nerve Palsy

<table>
<thead>
<tr>
<th>Location</th>
<th>Etiologies</th>
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<tr>
<td>Midbrain (nuclear and fascicular)</td>
<td>Aplasia of the nucleus, arteriovenous malformations or other vascular lesions, demyelination, hemorrhage (Raghavendra et al 2010; Sudhakar and Bapuraj 2010), ischemia or infarction (Keane 2004; Choi et al 2010), and trauma (Prost and Majetschak 2007), including surgical injury (Klaingutti et al 2004).</td>
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<tr>
<td>Subarachnoid space</td>
<td>Aneurysms (eg, superior cerebellar artery), pseudotumor cerebri (Speer et al 1999), increased intracranial pressure and hydrocephalus, inflammation (eg, polyarteritis nodosa) (Shimohata et al 2003; Borruat et al 2005), infections (eg, mastoiditis, meningitis, Lyme neuroborreliosis (Bababeygy and Quiros 2011)), percutaneous balloon (Urculo et al 2004), shunt procedure (Pandey et al 2001a; Giesemann et al 2012), halo traction (Pinches et al 2004), anterior temporal lobectomy (Cohen-Gadol et al 2003), post-lumbar puncture or spinal anesthesia, trauma, neoplasm (eg, carcinomatous meningitis, cerebellar hemangioblastoma, ependymoma, meningioma, metastasis, neurilemmoma, pineal tumors, schwannoma, trochlear nerve sheath tumors, or germinoma) (Shenouda et al 2002; Ture et al 2002; Du et al 2003; Shenoy and Raja 2004; Surucu et al 2007; Petrela et al 2009; Akle and Duprez 2009; Elmaleh et al 2009; Elfein et al 2010; Lima et al 2011; Young et al 2012), basilar artery dolichoectasia (Kawasaki and Purvin 2006), intracranial dermoid cyst (Tailor et al 2009), Lemierre syndrome (Lee et al 2009), and dural arteriovenous fistula (Mariniello et al 2012).</td>
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<tr>
<td>Cavernous sinus</td>
<td>Neoplasm, such as meningioma, Rathke cleft cyst (Park et al 2004) and pituitary adenoma (Kim et al 2007); infection, such as syphilis, tuberculosis, herpes zoster (Tsuda et al 2007); inflammation, such as sarcoid or the Tolosa-Hunt syndrome (Tsuda et al 2012); or vascular lesions, such as carotid-cavernous fistulas, aneurysm (Hall et al 2002).</td>
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<tr>
<td>Orbital lesion</td>
<td>Neoplasm; thyroid ophthalmopathy may mimic (Moster et al 1992), infection, infiltration, inflammation, or trauma; botulinum toxin injection (Kothari et al 2012).</td>
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The topographical anatomy of the fourth cranial nerve includes a nuclear, fascicular, subarachnoid space, cavernous sinus, superior orbital fissure, and orbital portion. The nerve begins at the fourth nerve nucleus and is located in the midbrain at the level of the inferior colliculus. The fourth cranial nerve is the only cranial nerve with a dorsal exit from the brainstem and has the longest intracranial course. The nerve crosses in the anterior medullary velum, passes between the superior cerebellar artery and the posterior cerebral artery, and continues into the subarachnoid space. The nerve travels within the lateral wall of the cavernous sinus and enters the orbit via the superior orbital fissure to innervate the superior oblique muscle. Newer and more detailed MRI studies of the orbital anatomy of patients with fourth cranial nerve palsy (congenital and acquired from multiple etiologies) have shown atrophy of the ipsilateral superior oblique muscle (Kono and Demer 2003; Uchiyama et al 2010) and enlargement of the contralateral inferior rectus muscle (Jiang and Demer 2008). Demer has proposed that the extraocular muscles pass through fibromuscular connective tissue pulleys that stabilize the muscle paths and control the direction of muscle pull (Demer 2006). Using coronal MRI, they also showed significantly reduced superior oblique cross-sectional areas and lack of contractile changes with vertical gaze in subjects with superior oblique palsies. Compared with normal control orbits, orbits with superior oblique palsies showed a statistically significant superior displacement of the medial rectus pulley (Clark and
Epidemiology

Fourth nerve palsy may affect patients of any age, any race, and either gender.

Prevention

There are no preventive measures to avoid fourth nerve palsy.

Differential diagnosis

The differential diagnosis includes thyroid-related orbitopathy, myasthenia gravis, and skew deviation (ocular tilt reaction). Less commonly, orbital floor fracture, orbital tumor, idiopathic orbital inflammatory syndrome, partial third nerve palsy, and synostotic plagiocephaly may simulate a fourth nerve palsy (Tamhankar et al 2011b; Matalia et al 2013). These entities can usually be distinguished on clinical grounds. Patients with thyroid-related orbitopathy or other restrictive extraocular myopathies usually demonstrate other orbital signs, such as lid lag, proptosis, chemosis, or conjunctival injection, and positive forced ductions. Rarely, patients with thyroid-related orbitopathy may present with an isolated hypertropia without other orbital signs (Moster et al 1992). Some of these patients may have a positive 3-step test. Orbital imaging (MRI, CT, or ultrasound) will often show enlargement of the extraocular muscles. Myasthenia gravis may rarely mimic a fourth nerve palsy (Rush and Shafrin 1982). Usually, other signs of myasthenia such as ptosis, variability, weak lid closure, and fatigue are present. Skew deviation (ocular tilt reaction) is a vertical misalignment owing to asymmetric disruption of the utriculo-ocular pathway and is usually accompanied by other brainstem signs. However, skew deviation may also mimic an isolated fourth nerve palsy (Donahue et al 1999; Wong 2010). Skew deviation can be distinguished from fourth nerve palsy by examining ocular torsion. Skew deviation is often associated with extorsion of the hypotropic eye and intorsion of the hypertropic eye in distinction to the ipsilateral extorsion that occurs in the hypertropic eye in fourth nerve palsy (Pandey et al 2008b). Examining ocular torsion has been proposed as the “fourth-step” to enable the differentiation of skew deviation from fourth nerve palsy (Donahue et al 1999). The “upright-supine test” also enables differentiation of skew deviation from fourth nerve palsy (Wong 2010; Wong et al 2011). When the head is placed in the supine position, the asymmetric utriculo-ocular pathway output that causes skew deviation is nullified. Therefore, placing the head in the supine position reduces the vertical misalignment of patients with skew deviation, a phenomenon that does not occur in fourth nerve palsy (Wong et al 2011).

Diagnostic workup

Fourth nerve palsies may be divided into those that are isolated and those that are accompanied by other contributory neurologic abnormalities (nonisolated). The evaluation of isolated fourth nerve palsies is determined by the clinical setting. The most common etiologies of isolated fourth nerve palsy are congenital (38%), traumatic (29%), microvascular (23%), and idiopathic (8%). Tumors are a rare cause (less than 1%) (Mollan et al 2009). Traumatic, congenital, and microvascular palsies do not typically require additional neuroimaging.

Patients with congenital fourth nerve palsies may present in childhood or as adults with decompensation of the ability to control their ocular misalignment. Congenital fourth nerve palsy is diagnosed by establishing evidence of chronicity on history and physical examination. Examination of old photographs may show a head tilt as a child, years before the onset of diplopia. Facial asymmetry with hypoplasia on the side of the head tilt is evidence that the head tilt has been present since childhood. Patients with longstanding fourth nerve palsy develop sensory adaptations to their extorsion of the involved eye and show minimal subjective torsion on double Maddox rod testing, despite the presence of anatomic extorsion on fundus examination. Finally, patients with congenital fourth nerve palsy often have increased vertical fusional amplitudes, often greater than 10 prism diopters. In contrast, patients with acquired, acute fourth nerve palsy are able to fuse only 2 to 3 prism dipters of vertical misalignment. Rarely, patients with fourth nerve palsy from slow-growing tumors may also show increased vertical fusional amplitudes (Petermann and Newman 1999). Nonetheless, patients with isolated fourth nerve palsy and signs of chronicity likely have a congenital fourth nerve palsy and do not require further diagnostic evaluation.

Patients with traumatic fourth nerve palsy will have a history of significant head trauma. Neuroimaging is typically obtained at the time of the trauma, and no further diagnostic evaluation is required if the history and physical findings
are consistent with the trauma.

Acquired acute fourth nerve palsies are often caused by ischemia to the subarachnoid segment of the nerve. An ischemic cause can be presumed if the fourth nerve palsy meets the following criteria:

- Isolated with no other focal neurologic signs
- Patient has arteriosclerotic risk factors such as diabetes mellitus, hypercholesterolemia, tobacco use, hypertension, older than 50 years of age
- No history of cancer
- The motility deficit remains isolated during follow-up examinations
- The fourth nerve palsy stabilizes after 1 to 2 weeks and resolves in 3 to 4 months

Patients who fulfill these criteria do not require neuroimaging studies at the outset (Miller et al 1999). They may be observed for improvement over the ensuing 3 to 4 months. Spontaneous recovery occurs within 12 weeks in more than 95% of cases. Patients with progressive or unresolved palsies, new neurologic signs or symptoms, and those without resolution of the palsy after several months should undergo neuroimaging. In a prospective review of imaging for acute third, fourth, or sixth cranial mononeuropathies that fulfilled the ischemic criteria noted above (Murchison et al 2011), 4 of 93 patients had lesions on MRI. All 4 of these patients had sixth cranial nerve palsy, and in only 1 of these 4 patients was the lesion related to the sixth cranial nerve dysfunction. The lesions detected were incidental in the other 3 patients. None of the 27 patients with fourth cranial nerve palsy in this study had lesions on MRI. The total modeled cost of imaging for these 93 patients was $1,688 to determine an underlying cause in 1 patient who had no subsequent change in treatment. Their study validates the traditional approach of not obtaining neuroimaging in patients with acute ocular motor cranial nerve palsies that meet the criteria described above.

Another study found a similarly low incidence of nonischemic isolated fourth nerve palsy, but suggested imaging (Chou and et al 2004). That study prospectively evaluated 66 patients older than 50 years of age (median 67 years, range 50 to 85 years) with acute isolated third, fourth, or sixth cranial mononeuropathies that fulfilled the ischemic criteria noted above. Nonischemic causes were identified by neuroimaging in 14 (21%), including neoplasm, brainstem infarcts, aneurysm, demyelinating disease, and pituitary apoplexy. In that study, 14 of the 66 patients had isolated fourth nerve palsy. Thirteen were due to peripheral microvascular disease; only 1 had a tumor as the cause of the fourth cranial nerve palsy diagnosed by neuroimaging. Further details regarding this case are not described. The authors concluded that “neuroimaging may have a role in the initial evaluation of adults with acute ocular motor mononeuropathies, regardless of age.” A third study prospectively evaluated 80 patients with acute isolated ocular motor nerve palsies that fulfilled the ischemic criteria noted above (Tamhankar et al 2013). Neuroimaging disclosed nonischemic causes in 5 patients, including pituitary apoplexy, a cavernous sinus B-cell lymphoma, and a petroclival meningioma. But among 22 patients with fourth cranial nerve palsy, neuroimaging found only 1 patient with a cause other than extra-axial nerve ischemia—a dorsal midbrain infarct. The authors recommended imaging in the initial evaluation of patients with isolated ocular motor nerve palsies, but presumably on the basis of the findings applied to sixth and third nerve palsies, not fourth nerve palsies.

Elderly patients who present with headache, scalp tenderness, jaw claudication, or visual loss, and an ophthalmoplegia or diplopia, should undergo an appropriate evaluation for giant cell arteritis, including erythrocyte sedimentation rate and temporal artery biopsy (Reich et al 1990). Patients with variable or fatigable motility findings or ptosis should be evaluated for myasthenia gravis.

Patients with acute isolated fourth nerve palsies who are young or who lack arteriosclerotic risk factors could undergo prompt neuroimaging, but observation for spontaneous improvement is also justified because the palsies usually resolve and have no apparent cause (Galetta and Balcer 1998; Feinberg and Newman 1999; Petermann and Newman 1999). Two retrospective case series with follow-up of greater than 6 months described the prognosis of isolated, idiopathic, fourth cranial nerve palsies. One study reported that 12 of 15 cases had resolved by 4 months after a mean follow-up of 5.5 years (Coppeto and Lessell 1978). Another study described 13 cases with follow-up ranging from 4 to 7 years; all had resolved within 10 weeks (Nemet et al 1980). None of the patients in either series developed new neurologic manifestations over an extensive follow-up period. Patients without improvement after 3 months, however, should undergo neuroimaging. Cerebral angiography is not recommended unless an aneurysm is suggested by other neuroimaging studies. One report described isolated fourth cranial nerve palsies due to superior cerebellar aneurysms, but both patients described headaches (Agostinis et al 1992). In these cases, noninvasive neuroimaging studies
confirmed the presence of the aneurysm prior to digital angiography. All patients with progressive or unremitting deficits should be considered for lumbar puncture if neuroimaging is normal. Patients with nontraumatic bilateral fourth nerve palsies should undergo neuroimaging and be considered for lumbar puncture if neuroimaging is normal.

Nonisolated palsies may be divided according to the location of the lesion: (1) midbrain (nucleus fascicle syndrome); (2) subarachnoid space; (3) cavernous sinus; and (4) orbit (Brazis 2009; Prasad and Volpe 2010).

A midbrain lesion causing fourth nerve palsy will manifest hemisensory loss, hemiparesis, a contralateral relative afferent pupillary defect (Taguchi et al 2000), Horner syndrome (Gold et al 2012), or other cranial neuropathies. The differential diagnosis includes midbrain ischemia, hemorrhage, demyelination, and neoplasm. A brainstem lesion, often traumatic, is suggested when fourth nerve palsies are bilateral and simultaneous (Bhola and Olson 2006).

Although lesions of the subarachnoid space may be associated with isolated fourth nerve palsy, other manifestations are usually present, including headache, stiff neck, and other cranial neuropathies.

Cavernous sinus lesions are usually associated with third, fifth, or sixth nerve palsies or Horner syndrome. One report described a fourth nerve palsy due to a posterior draining carotid-cavernous fistula (Tsai et al 2000).

Typically, orbital lesions produce signs such as proptosis, chemosis, and orbital or conjunctival edema. Neuroimaging should be directed to the orbit. This is one report of superior facial squamous cell carcinoma with perineural spread to the superior oblique muscle belly (Wilcsek et al 2000).

Magnetic resonance imaging might show a small superior oblique muscle owing to denervation atrophy (Gore and Malik 2006; Demer 2010). Congenital superior oblique palsy may be associated with absence of the trochlear nerve in addition to superior oblique muscle atrophy (Yang et al 2012).

**Management**

Acute isolated ischemic fourth nerve palsies may be observed for improvement. Patching 1 eye will alleviate binocular diplopia and is useful for patients who are being observed or who defer to prisms or surgery. Spectacle prisms may be employed, but the degree of inconstant misalignment is often an impediment to obtaining a wide zone of single binocular vision (Tamhankar et al 2011b). Strabismus surgery may be helpful in patients failing spectacle prisms (Farvardin and Nazarpour 2002; Mellott et al 2002; Bahl et al 2013).

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Former authors
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ICD and OMIM codes

ICD codes
ICD-9:
Isolated fourth nerve palsy: 378.53

ICD-10:
Fourth [trochlear] nerve palsy: H49.1

Profile

Age range of presentation
0-01 month
01-23 months
02-05 years
06-12 years
13-18 years
19-44 years
45-64 years
65+ years

Sex preponderance
male=female

Family history
none

Heredity
none

Population groups selectively affected
none selectively affected

Occupation groups selectively affected
none selectively affected

Differential diagnosis list
thyroid related orbitopathy
skew deviation (ocular tilt reaction)
myasthenia gravis
restrictive vertical strabismus
orbital floor fracture
orbital tumor
orbital pseudotumor
other paretic vertical strabismus
partial third nerve palsy
Other topics to consider

Combined third, fourth, and sixth nerve palsies
Isolated third nerve palsy
Isolated sixth nerve palsy