PSS NEWS

An On-Line Publication for COPE Continuing Education in Optometry

Abducens Nerve Palsies

2.0 Hours - COPE 69959- NO

This program is supported by an educational grant from Alcon

Leonid Skorin Jr, OD, DO, FAAO, FAOCO

Learning Objectives:

1. *To correlate the anatomical course of the abducens cranial nerves with a patient's clinical presentation*
2. *To differentiate between abducens nerve palsies and their imposters*
3. *To determine an appropriate treatment regimen for a patient with an isolated abducens nerve palsy*

Case Presentation

An 83-year-old female presents with a recent onset horizontal diplopia. She also complains of occipital headaches, fatigue and one recent episode of amaurosis fugax. The patient denied being a diabetic and she felt her hypertension was adequately controlled with atenolol. She does take one 81 mg. aspirin daily and has been recently taking acetaminophen for her occipital headaches without relief.

Examination confirmed a left abducens cranial nerve palsy. There was no afferent pupillary defect, visual acuity was 20/25 best corrected in each eye (the patient had mild-moderate nuclear sclerotic cataracts), there was no papilledema and no other neurologic findings were present.

Based on the patient's history and examination findings a tentative diagnosis of either vasculopathic mononeuropathy or temporal arteritis was made. The patient's blood pressure was measured in the office and found to be normal. Blood work testing found a normal fasting blood sugar but the patient's complete blood count was positive for normochromic, normocytic anemia. Her Westergren erythrocyte sedimentation rate was grossly elevated at 120mm/hr and her C-reactive protein value was also elevated.

The final diagnosis was temporal arteritis which was confirmed by temporal artery biopsy. The patient was started on high dose oral prednisone and showed a significant improvement in all her signs and symptoms.

Discussion of Case Presentation

This case highlights the importance of not only localizing the abducens cranial nerve palsy but incorporating this finding within the context of the patient's other neurologic signs and symptoms. Although this patient had definite risk factors for vasculopathic disease (advanced age and hypertension) it was

actually her other neurologic (occipital headaches, amaurosis fugax) and constitutional (fatigue) symptoms in combination with the recent onset diplopia that helped point to another possible vision threatening diagnosis: temporal arteritis.

Further evaluation helped confirm this condition:

- Blood pressure measurement confirmed that the patient's hypertension was medically controlled.
- Diabetes was ruled-out with a fasting blood sugar.
- Anemia was identified with a complete blood count. Low-grade anemia is a common finding in patients with temporal arteritis and helps explain the patient's complaint of fatigue.
- The erythrocyte sedimentation rate and the serum C-reactive protein were elevated. There is a combined sensitivity of diagnosing temporal arteritis of 97% when both these tests are elevated.

ANATOMY

The paired sixth (abducens) cranial nerve nuclei are located in the brainstem at the level of the pons, in the caudal portion of the paramedian pontine tegmentum, beneath the floor of the fourth ventricle.[1] Each nucleus contains two types of neurons: large motor cells whose axons form the ipsilateral sixth cranial fascicle and nerve, and a group of smaller cells, the internuclear neurons. The internuclear neurons send their axons across the midline to travel in the contralateral medial longitudinal fasciculus (MLF), where they synapse in the contralateral medial rectus subnucleus to coordinate conjugate horizontal gaze.[2] The seventh (facial) cranial nerve fibers loop around the abducens nuclei. The abducens fascicle travels anteriorly through the pons, lateral to the parapontine reticular formation, and through the pyramidal tract to exit the brain stem into the subarachnoid spaces within the cerebellopontine angle.[3] It then travels up the clivus over the petrous ridge where the nerve fibers are anchored at the base of the skull to the tips of the petrous pyramids by the petrosphenoidal ligaments. The nerve fibers enter the substance of the cavernous sinus and lie laterally, adjacent to the carotid arteries. The nerve fibers enter the orbit through the medial portion of the superior orbital fissure where they innervate the lateral rectus muscle which moves the eye laterally.[4]

The patient's clinical presentation is a direct result of the location of the lesion along the long and complicated course of the abducens nerve. Any associated neurologic signs or symptoms are a direct consequence of the local neuroanatomy.

CONGENITAL ABDUCENS NERVE PALSIES

MOBIUS SYNDROME (CONGENITAL BULBAR PARALYSIS)

Mobius syndrome is the occurrence of congenital bilateral sixth nerve and facial nerve deficits because the nerves, nuclei or both do not develop.[5] The following are common clinical findings: patients have a mask-like facies, the mouth is often held open, atrophy of the tongue indicates hypoglossal cranial nerve involvement, the eyelids cannot be closed completely, deafness and mental retardation may occur, and there is complete loss of horizontal eye movement but vertical eye movements are usually normal.[5,6] Up to 40% of these children have an esotropia that is usually 50 prism diopters or greater.[7]

DUANE'S RETRACTION SYNDROME

There are three types of Duane's retraction syndromes. Type I is the form that can be

confused with congenital esotropia or an abducens nerve palsy. This disorder of ocular motility is characterized by limitation of abduction, narrowing of the palpebral fissure and retraction of the globe on adduction, slight or no limitation of adduction and upshoot or downshoot on adduction.[8] There is a female preponderance and a predilection for the left eye. The exact etiology for this syndrome is not known. Numerous theories including mechanical, innervational and central nervous system anomalies have all been implicated.

ABDUCTION DEFECTS NOT DUE TO ABDUCENS PALSY

Not all abduction deficits are actual sixth nerve palsies. Various orbital conditions such as muscle entrapment secondary to trauma or sinus surgery or dysthyroid ophthalmopathy which causes a restriction of the medial rectus muscle can cause an abduction deficit. Forced duction testing would identify a restricted muscle when the globe is rotated manually in the direction of the limited duction.

Other causes of abduction deficits include inflammatory orbital pseudotumor (myositis) and ocular myasthenia gravis. The Tensilon (intravenous edrophonium) test would show temporary resolution of the abduction deficit indicating myasthenia as the underlying cause.

Spasm of the near reflex can simulate bilateral sixth nerve palsies. Pupillary miosis, which is part of the near triad along with convergence and accommodation helps confirm the diagnosis. Occluding one of the patent's eyes will often resolve the abduction deficit including the miosis and pseudomyopia.[4]

It should be remembered that a break in fusion of a congenital esophoria can mimic an abduction paresis.

ACQUIRED ABDUCENS NERVE PALSIES

Abducens nerve palsies often present with other neurologic or neuro-ophthalmic findings that help localize the lesion. Lesions that damage the abducens nucleus produce a conjugate gaze palsy to the ipsilateral side. Involvement of the adjacent paramedian pontine reticular formation and medial longitudinal fasciculus can produce the one-and-a-half syndrome which consists of a horizontal gaze palsy combined with an internuclear ophthalmoplegia.[6]

Lesions that involve the abducens nerve fascicles produce an ipsilateral abducens nerve palsy and also affect adjacent pontine structures. Lesions in the pontine tegmentum may produce an ipsilateral facial palsy, Horner's syndrome, facial anesthesia and peripheral deafness. This syndrome of the dorsal pons is known as Foville's syndrome. Ventral pontine lesions involving the abducens fascicles also involve the corticospinal tracts and facial nerve fascicles. Such lesions produce an ipsilateral abducens palsy with contralateral hemiparesis. If there is an accompanying ipsilateral facial cranial nerve palsy this is known as Millard-Gubler syndrome and if there is no facial nerve palsy, it is known as Raymond-Cestan syndrome.[6]

Within the subarachnoid space, the abducens nerve travels a long intracranial course. Although it was thought that this extensive length made the nerve more vulnerable to numerous insults, it is actually the location and course, rather than its length, that are the major factors that lead to an abducens nerve paresis.[6] In this location the nerve lies in close proximity or is actually bound to various blood vessels. These include the anterior inferior cerebellar artery, posterior inferior cerebellar artery and the basilar artery. Any of these blood vessels can cause compression of the nerve as they become atherosclerotic or from aneurysmal formation.[9]

Elevated intracranial pressure may result in downward displacement of the brainstem, with stretching of the abducens nerve, which is tethered at its exit from the pons and in Dorello's canal found at the tip of the temporal bone of the skull. This downward

and forward displacement can result in up to 30% of pseudotumor cerebri patients exhibiting either unilateral or bilateral abducens nerve palsies.[10]

Inflammation of the petrous part of the temporal bone and its dura may occur secondary to suppurative otitis media or mastoiditis causing an abducens nerve palsy, facial pain from trigeminal ganglion involvement, facial paralysis from facial nerve involvement and decreased hearing from vestibulocochlear nerve involvement. These signs and symptoms comprise the petrous apex syndrome or Gradenigo's syndrome.[1,2,6] With today's use of antibiotics most cases of this syndrome are due to tumors and aneurysms of the intrapetrosal segment of the internal carotid artery.[6]

A lesion located more anteriorly in the sphenopalatine fascia would involve the sphenopalatine nerve and the second division (maxillary) of the trigeminal nerve, causing an abducens nerve palsy accompanied by loss of tearing, parasthesias or pain involving the lower two-thirds of the face and ocular irritation ipsilateral to the paresis.[10] This is often caused by a nasopharyngeal carcinoma that has extended through foramina at the base of the skull and has spread beneath the dura to damage the extradural portions of the abducens and trigeminal nerves.[10] These signs and symptoms are known as pseudo-Gradenigo's syndrome.[11]

In the cavernous sinus, the abducens nerve is joined by the oculomotor, trochlear and the first division (ophthalmic) of the trigeminal cranial nerves. The oculo-sympathetic fibers of the carotid plexus are also found in the cavernous sinus. The abducens cranial nerve runs through the middle of the cavernous sinus, making the nerve more vulnerable to injury and often the first cranial nerve to be affected by cavernous sinus disease processes.[2] Examples of conditions causing abducens nerve involvement include expansile processes (internal carotid artery aneurysm, pituitary tumor, sphenoid mucocele, metastatic tumors), vascular problems (carotid-cavernous thrombosis, carotid-cavernous fistula, dural arteriovenous fistula), ischemic problems (diabetes mellitus, hypertension, temporal arteritis, migraine) and infectious-inflammatory processes (sinusitis, mucormycosis, herpes zoster).[5]

Isolated abducens nerve palsy accompanied by ipsilateral postganglionic Horner's syndrome, also points to a cavernous sinus lesion. This presentation can occur in association with primary and traumatic intracavernous aneurysms and with both benign and malignant tumors that arise in or invade the cavernous sinus[6].

Painful ophthalmoplegia consisting of periorbital or hemicranial pain combined with ipsilateral cranial nerve palsy, oculo-sympathetic paralysis and sensory loss in the distribution of the ophthalmic and occasionally the maxillary division of the trigeminal nerve is known as Tolosa-Hunt syndrome.[12] Various combinations of these cranial nerve palsies may occur, localizing the pathologic process to the region of the cavernous sinus or superior orbital fissure. Lesions that occur more anteriorly at the orbital apex and superior orbital fissure are difficult to differentiate clinically from cavernous sinus lesions unless they are accompanied by proptosis and optic neuropathy.

ISOLATED ABDUCENS NERVE PALSIES

Abducens nerve palsies occur more frequently than do palsies of the oculomotor or trochlear cranial nerve.[13] The most frequent cause of isolated abducens nerve palsy in individuals older than 50 years of age is infarction of the nerve trunk also known as ischemic mononeuropathy.[2,4] In one large series of cases, microvascular infarction was due to either diabetes mellitus, hypertension, atherosclerosis or a combination of these conditions.[14] These patients often complain of

retrobulbar or periocular pain, which may begin up to one week before the palsy onset. Paresis in these patients presents in an acute fashion and resolves, or at least improves, spontaneously within three months. Reexamination of these patients in the first two to four weeks after onset is important to confirm that the pattern is remaining isolated and not progressing or involving other cranial nerves.[2]

Temporal arteritis is a granulomatous vasculitis that affects large and medium diameter vessels with a predilection for arteries of the head and neck. The incidence of ophthalmoplegia and temporal arteritis has been estimated to be between 5% and 15%.[15] The cause of this ophthalmoplegia is ischemia to the extraocular muscle, secondary to vasculitic occlusion of the muscular branches of the ophthalmic artery.[16] This muscle palsy may be transient. Complete resolution of an isolated abducens nerve palsy in two or three weeks rather than two or three months may be a clinical clue that temporal arteritis is the basis of the problem rather than atherosclerotic, small vessel disease.[15]

If untreated, many patients go on to lose vision. In a series of 22 patients with diplopia secondary to temporal arteritis from the Mayo Clinic, ten later lost vision in one or both eyes, while six others had retinal ischemic episodes.[16] The standard steroid treatment for palsy secondary to temporal arteritis has been shown to resolve the ophthalmoplegia.[17]

Neoplasms causing compression of the abducens nerve are characterized by progressive worsening of the abduction deficit, often with the development of other associated cranial neuropathies or evolving neurologic signs.[2] Magnetic resonance imaging with contrast dye enhancement is indicated in patients whose horizontal diplopia is slowly progressing, in patients with a history of carcinoma, if other cranial nerves are involved, if the patient has unrelenting facial pain or if other neurologic symptoms are present.[2,4] If suspicion points to specific sinus or mastoid disease, computed tomography can be used to better delineate these entities.[3] Any local or metastatic tumor can be the underlying cause but of neoplasms involving the cerebellopontine angle, 90% are acoustic neuromas with the remaining 10% being meningiomas or cholesteatomas.[18]

Children and young adults with isolated abducens nerve palsy are more likely to have a serious, identifiable underlying cause. Up to 39% of such deficits in children were found to result from primary brain stem gliomas or cerebellar tumors.[19] Unfortunately, the prognosis for these children is usually poor since these tumors have a tendency to spread by infiltration, causing multiple cranial nerve deficits, pyramidal tract dysfunction and ataxia.[20] In those children who do not have a neoplasm, a post-viral syndrome or recent immunization may be the underlying cause of an isolated abducens palsy.[21,22] These palsies resolve within four months but may recur and eventually leave the patient with a permanent abduction deficit.[22,23]

Other causes of isolated abducens nerve palsies include trauma, demyelinating disease (multiple sclerosis), sarcoidosis, mastoiditis, meningitis, migraine, post-lumbar puncture, stroke, pseudotumor cerebri or other causes of elevated intracranial pressure, myelography and Wernicke's encephalopathy which is associated with chronic alcoholism.[4,14,18]

Elevated intracranial pressure either from pseudotumor cerebri or neoplasm, lumbar myelography, lumbar puncture and Wernicke's encephalopathy can all cause unilateral or bilateral abducens nerve palsies.[24,25]

Wernicke's encephalopathy may include several eye signs, among which are nystagmus of a nonspecific character, horizontal gaze paralysis and rarely vertical gaze paralysis with the abducens nerve palsy. Gait ataxia and somnolence make up the other classic

findings of this condition. If the patient also exhibits confabulation then the patient would actually have the more complicated Wernicke-Korsakoff syndrome.[18]

The abducens nerve palsies seen after spinal procedures (lumbar puncture, spinal anesthesia, myelography or shunting) generally have been considered to be caused by chronic leakage of spinal fluid from the tap site with displacement of the brain and traction on pain-sensitive structures (post-spinal-tap headache) and traction on the abducens nerve with compression over firm structures such as the petrous bone.[18] Significant features of this syndrome are the delayed appearance of the paresis (5 to 14 days after the spinal procedure) and the invariable recovery, usually within four to six weeks.[6] If no other neurologic findings are present, no further investigation is required and no treatment is necessary.

CLINICAL EVALUATION OF ABDUCENS NERVE PALSY

The most common complaint herd from patients with isolated abducens nerve palsy is that of horizontal diplopia, which is eliminated when one eye is closed. This horizontal diplopia is worse at distance than near and is exaggerated when the patient looks in the direction of the affected lateral rectus muscle.[13]

A thorough neurologic assessment, particularly with respect to the first eight cranial nerves and the integrity of the oculosympathetic pathway must be performed.[6] Clinical evaluation should identify only an abduction deficit in a truly isolated abducens nerve palsy. Cover testing indicates esotropia, which is greater when the paretic eye is fixing a distant target, compared to when the unaffected eye is fixing on the same target. If duction testing finds the paretic eye abducting farther than when versions are evaluated, a neurogenic abducens nerve palsy can further be confirmed (Figure 4). The paretic eye will also show hypometric saccades and optokinetic drum or tape examination will disclose asymmetric refixation movements. Finally, forced duction testing will help differentiate between a neurogenic and mechanical restriction, which is seen when there is infiltration or entrapment of the lateral rectus muscle.

Ocular assessment should also include corneal sensitivity testing with a cotton wisp or aesthesiometer. The most sensitive part of the trigeminal nerve appears to be those fibers subserving the corneal reflex. The earliest sign of trigeminal cranial nerve damage is often an impaired or absent corneal response. Presence of a decreased or absent corneal reflex, facial paresis, hearing loss, Horner's syndrome, proptosis or papilledema all warrant further neuro-ophthalmic imaging studies.

Children who present with a recent history of a flu-like syndrome or vaccination may be observed without performing neuroimaging. Initially, the child should be examined every two weeks to determine if there is any progression of the paresis or for the development of additional neurologic problems. If there is no improvement after four months then neuroimaging is indicated.[4]

In adults younger than 50, magnetic resonance imaging following the course of the abducens nerve is necessary. Even when the palsy is isolated, this age group is more likely to have a serious, identifiable underlying cause.[26] These may include a vasculopathic ischemic mononeuropathy from diabetes or hypertension, collagen vascular disease, Lyme disease, syphilis, demyelinting disease such as multiple sclerosis and neoplasm.

Since the most frequent cause of isolated abducens palsy in patients over age 50 is ischemic mononeuropathy, these individuals need to have their blood pressure measured and a blood workup done.[2,4] The blood testing should include a complete blood count with

differential (to rule-out anemia or leukemia) and a fasting blood sugar (to rule-out diabetes). There is no correlation between severity of glucose metabolism defect and occurrence of cranial nerve palsies, thus, an isolated abducens nerve palsy may signal the presence of otherwise occult diabetes. The diabetic cranial neuropathies appear to be related more to the length of time the person has had diabetes.

In more elderly individuals where the clinician may suspect temporal arteritis, an erythrocyte sedimentation rate and C-reactive protein should be performed.

All patients with a suspected ischemic mononeuropathy nerve deficit need to be monitored monthly. If no change in the cranial nerve palsy occurs over a three-month period, if it becomes worse, or if new signs or symptoms appear, neuroimaging needs to be done.[4,6,11]

MANAGEMENT OF ABDUCENS NERVE PALSY

In an acute phase of an abducens cranial nerve palsy simple occlusion of the paretic eye with patching or an opaque contact lens is all that may be necessary to relieve the patients diplopia.[5,13] Patients under eight years old should undergo alternate patching of the eyes to prevent amblyopia.[6] If the deviation is small and not greatly incomitant, temporary prism correction such as Fresnal prisms can be used.[13] Prism power must be monitored carefully, making appropriate power adjustments as the eye muscle imbalance resolves. Larger strabismic deviations are usually not amenable to prism correction.

Chemodenervation using botulinum toxin may be necessary if the recovery is prolonged or the deviation is too large for prism correction. The ipsilateral medial rectus muscle is injected with the botulinum toxin to prevent contracture of the antagonist muscle while waiting for recovery of the involved abducens cranial nerve. The initial injection consists of one to three units of toxin, just enough to weaken the medial rectus but not enough to cause prolonged dysfunction.[3] Chemodenervation can be used in both acute and chronic abducens nerve palsies. It works best in chronic palsies if there is some remaining lateral rectus function.[3]

Surgical correction can be done after measurements have been stable for at least six months.[13] Some surgeons recommend waiting at least eight to ten months before doing strabismus surgery.[6] Surgery, when required, usually consists of either weakening of the ipsilateral medial rectus muscle combined with strengthening of the ipsilateral lateral rectus muscle or some type of vertical muscle transposition procedure, often combined with chemodenervation of the ipsilateral medial rectus muscle.[6]

CONTINUING EDUCATION QUIZ

This article is worth two (2.0) continuing education credits. This COPE-approved program is accredited by the University of Alabama at Birmingham College of Optometry.

Once you have registered for the course, you will be given an access code and go to www.flexiquiz.com where you will take the quiz. To earn credit, you must receive a grade of 70% or greater.

Please note that all 50 states have different rules and regulations concerning the acceptance of correspondence/internet continuing education. Please verify with your state board of optometry as to the amount of allowable hours and acceptable categories/topics in your particular state.

Continuing Education Quiz

1. Which of the following statements in regard to abducens cranial nerve nuclei is *false?*
    A. The nuclei are paired.
    B. They are located in the brainstem at the level of the pons.
    C. They are located above the floor of the third ventricle.
    D. Each nucleus contains two types of neurons.

2. After the abducens cranial nerve fibers enter the cavernous sinus they lie adjacent to which artery?
    A. Basilar.
    B. Vertebral.
    C. Cerebellar.
    D. Carotid.

3. The abducens cranial nerve innervates which extraocular muscle?
    A. Lateral rectus.
    B. Medial rectus.
    C. Superior oblique.
    D. Inferior oblique.

4. The abducens cranial nerve fibers enter the orbit through which anatomical structure?
    A. Optic foramen.
    B. Superior orbital fissure.
    C. Foramen rotundum.
    D. Foramen magnum

5. Which of the following is a common clinical finding of Mobius syndrome?
    A. Mask-like facies.
    B. Hypertrophy of the tongue.
    C. Blepharospasm.
    D. Inability to look up or down.

6. Which of the following statements in regard to Duane's retraction syndrome is *false*?
    A. It is more common in males.
    B. The left eye is affected more often.
    C. There are three different variants to this syndrome.
    D. It is a congenital disorder.

7. Type I Duane's retraction syndrome will show all the following findings *except*?
    A. Limitation of abduction.
    B. Widening of the palpebral fissure on adduction.
    C. Globe retraction on adduction.
    D. Upshoot or downshoot on adduction.

8. Which of the following may cause an abduction deficit?
    A. Dysthyroid ophthalmopathy.
    B. Inflammatory orbital pseudotumor.
    C. Ocular myasthenia gravis.
    D. All of the above.

9. Which of the following statements in regard to spasm of near reflex is *false*?
    A. It can simulate bilateral sixth nerve palsies.
    B. Pseudomyopia is a common finding.
    C. Pupillary mydriasis is a common finding.
    D. Occluding one of the patients eye will often resolve the abduction deficit.

10. Which of the following syndromes presents with an ipsilateral abducens and facial cranial nerve palsy with a contralateral hemiparesis?
    A. Raymond-Cestan.
    B. Millard-Gubler.

C. Gradenigo’s.

D. Foville’s

11. Which of the following syndromes consists of an ipsilateral conjugate gaze palsy and internuclear ophthalmoplegia?

    A. One-and-a-half.

    B. Foville’s.

    C. Pseudo-Gradenigo’s.

    D. Petrous apex.

12. Which of the following are common findings in Tolosa-Hunt syndrome?

    A. Painful ophthalmoplegia.

    B. Oculosympathetic paralysis.

    C. Sensory loss in the distribution of the ophthalmic division of the trigeminal nerve.

    D. All of the above.

13. The most frequent cause of isolated abducens nerve palsy in individuals older than 50 years of age is?

    A. Post-viral syndrome.

    B. Neoplasm.

    C. Ischemia.

    D. Trauma.

14. Which of the following statements in regard to temporal arteritis is *false*?

    A. It is a granulomatous vasculitis.

    B. The incidence of ophthalmoplegia and temporal arteritis has been estimated to be over 75%.

    C. The cause of the ophthalmoplegia is ischemia to the extraocular muscle.

    D. If untreated, many patients go on to lose vision.

15. Magnetic resonance imaging of the brain with contrast dye enhancement is indicated in which of the following cases?

    A. If the patient has unrelenting facial pain.

    B. If an abduction deficit is progressing.

    C. If other cranial nerves become involved.

D. All of the above.

16. What percentage of cerebellopontine tumors are acoustic neurons?

A. 10%.

B. 50%.

C. 75%.

D. 90%.

17. The Wernicke-Korsakoff syndrome is often the result of?

A. Neoplasm.

B. Spinal anesthesia.

C. Chronic alcoholism.

D. Demyelinating disease.

18. All of the following are common clinical findings of an isolated abducens nerve palsy *except*?

A. Horizontal diplopia worse at distance than near.

B. Diplopia persists when the paretic eye is occluded.

C. Diplopia is exaggerated when the patient looks in the directioon of the affected lateral rectus muscle.

D. Cover testing indicates esotropia.

19. Which of the following statements with regard to chemodenervation as a management tool for isolated abducens cranial nerve palsy is *false*?

A. Botulinum toxin is commonly used as a chemodenervating agent.

B. The ipsilateral lateral rectus muscle is injected.

C. The initial injection consists of one to three units of Botulinum toxin.

D. Chemodenervation can be used in both acute and chronic abducens nerve palsies.

20. Which of the following can be used in the management of isolated abducens cranial nerve palsy?

A. Fresnel prisms.

B. Opaque contact lens.

C. Strabismus surgery.

D. All of the above.

REFERENCES

1. Glaser JS, Bachynski B: Infranuclear disorders of eye movement, in Glaser JS (ed): Neuro-Ophthalmology. J.B. Lippincott Co., Philadelphia, 1990, pp. 361-418.
2. Martin TJ, Corbett JJ: Neuro-Ophthalmology, The Requisites in Ophthalmology. Mosby, St. Louis, 2000, pp. 154-160.
3. Siatkowski RM: Third, Fourth, and Sixth Nerve Palsies. Focal Points. Clinical Modules for Ophthalmologists. Vol. XIV, No. 8. American Academy of Ophthalmology, San Francisco, 1996, pp. 1-14.
4. Burde RM, Savino PJ, Trobe JD: Clinical Decisions in Neuro-Ophthalmology. 3rd ed. Mosby. St. Louis, 2002, pp. 171-183.
5. Newman NM: Neuro-Ophthalmology, A Practical Text. Appleton & Lange, Norwalk, CT, 1992, pp. 205-208.
6. Miller NR, Newman NJ: Walsh & Hoyt's Clinical Neuro-Ophthalmology, The Essentials. 5th Ed., Williams & Wilkins, Baltimore, 1999, pp. 509-561.
7. Nelson LB, Wagner RS, Simson JW, Harley RD. Congenital esotropia. Surv Ophthalmol 1987, 31: 363-383.
8. DeRespinis PA, Caputo AR, Wagner RS, Guo S: Duane's retraction syndrome. Surv Ophthalmol 1993; 38: 257-288.
9. McKinna AJ. Eye signs of 611 cases of posterior fossa aneurysm: their diagnostic and prognostic value. Can J Ophthalmol 1983; 92: 171-172,
10. Witte MC, Neel HB: Nasopharyngeal cancer, in Bailey BJ (ed): Head and Neck Surgery—Otolaryngology. 2nd ed. Vol. 2. Lippincott-Raven, Philadelphia, 1998, pp. 1637-1653.
11. Kline LB, Bajandas FJ: Neuro-Ophthalmology Review Manual. 4th ed., SLACK, Thorofare NJ, 1996, pp. 83-92.
12. Kline LB: The Tolosa-Hunt syndrome. Surv Ophthalmol 1982; 27: 79-95.
13. Skorin L: Abducens nerve palsy, in Onofrey BE, Skorin L, Holdeman NR (eds): Ocular Therapeutics Handbook: A Clinical Manual, Lippincott-Raven, Philadelphia, 1998, pp. 512-515.
14. Rush JA, Younge BR. Paralysis of cranial nerves III, IV and VI. Cause and prognosis in 1,000 cases. Arch Ophthalmol 1981; 99: 76-79.
15. Goodwin JA: Temporal Arteritis: Diagnosis and Management. Focal points. Clinical Modules for Ophthalmologists. Vol X, No. 2. American Academy of Ophthalmology, San Francisco, 1992, pp. 1-11.
16. Hollenhorst RW, Brown JR, Wagener HI, et al. Neurologic aspects of temporal arteritis. Neurology 1960; 10: 490-498.
17. Dimant J, Grob D, Brunner D. Ophthalmoplegia, ptosis and miosis in temporal arteritis. Neurology 1980; 30: 1054-1058.
18. Walsh TJ: Diplopia, in Walsh TJ (ed): Neuro-Ophthalmology: Clinical Signs and Symptoms. 3rd ed., Lea & Febiger. Philadelphia, 1992, pp. 124-175.