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Research Article

ANISOCORIA

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ABSTRACT

"Anisocoria means unequal pupils. One of the pupils is smaller or larger than the other. It may be congenital or acquired. Normally the pupils are equal in size under all light conditions. Few people can have small amount of anisocoria which is physiological. So, it's important to differentiate physiological anisocoria from pathological cause."

INTRODUCTION

Anisocoria means unequal pupils (iso – equal; coria – pupil). One of the pupils is smaller or larger than the other. It may be congenital or acquired. Normally the pupils are equal in size under all light conditions. If there is disparity in size of pupils, the cause can be:-

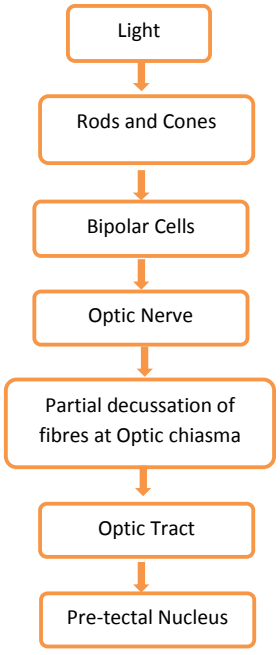
1. Injury to sphincter pupillae of Iris.
2. Injury to dilator pupillae of iris.
3. Defect in innervation to pupil.

Size of the pupil depends on various factors such as state of the iris, tone of sphincter and dilator muscles of the pupil, innervation to the pupillary muscles, events occurring at myo-neural junctions and general emotional state of the person.Few people can have small amount of anisocoria which is physiological and asymptomatic. So, it's important to differentiate physiological anisocoria from pathological cause. Physiological anisocoria means a normal supply by sympathetic and parasympathetic nervous system to both pupils, regardless of the surrounding lightening. In approximately 10% of the healthy individuals, there can be a difference in pupil size of 1 mm or less. Once, it has been confirmed that the anisocoria is not physiological; the next step is to find out which pupil is pathologically abnormal. The pupillary difference should be measured in both light and dark to determine the affected pupil.

Light Reflex Pathway

It is important to understand the normal afferent and efferent pathway of pupillary reflex for better understanding of the topic.

Afferent pathway



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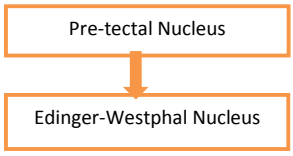
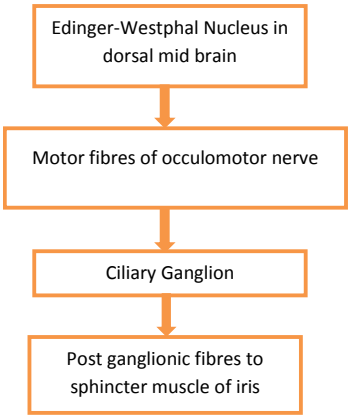
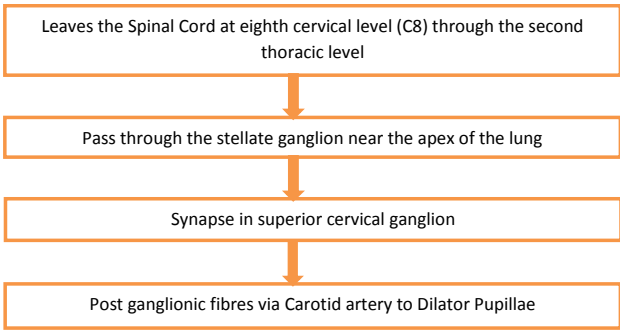
Inter-nuncial Fibres



Efferent pathway



Sympathetic Pathway



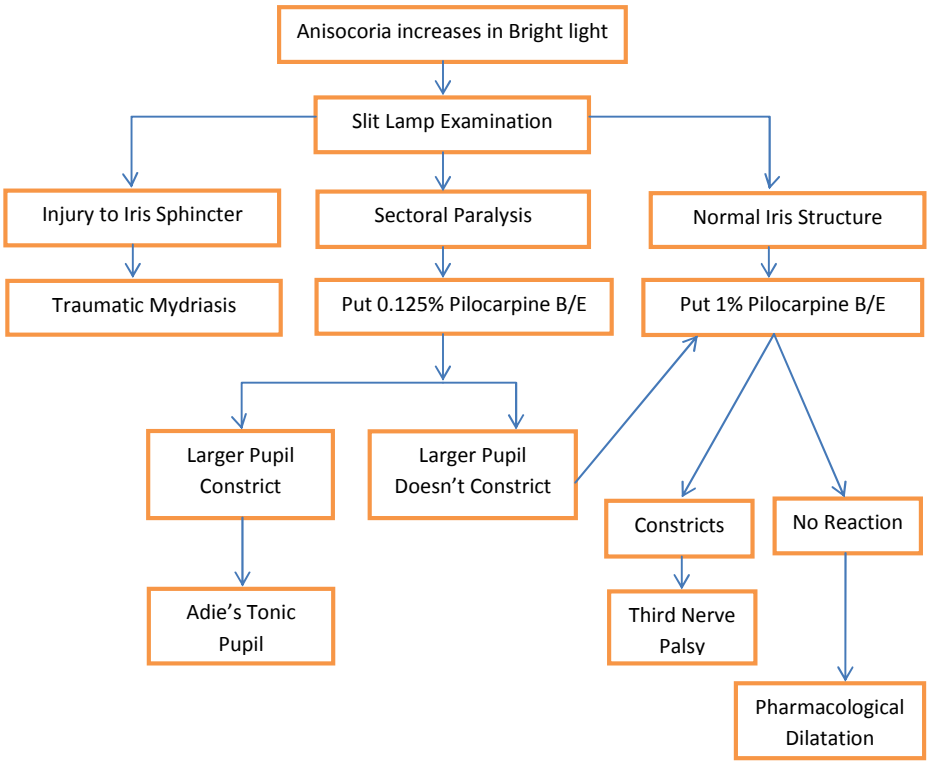
How to Approach

The first step is to find out the cause of anisocoria and the muscle which is not functioning adequately. Anisocoria always increases in the direction of iris muscle paralysed. If the sphincter pupillae is paralysed, the anisocoria will increase in bright light and if the dilator pupillae is paralysed, then the anisocoria will increase in dark. If the anisocoria is more prominent in the light, then the larger pupil is the abnormal one and the cause is an injury to ipsilateral parasympathetic supply. It is because of the fact that under bright light, both the pupils are stimulated by parasympathetic innervation to constrict. In case of parasympathetic injury, the ipsilateralabnormal pupil doesn't constrict and the contralateral pupil constricts briskly. In the dark, the sympathetic nervous system stimulates both the pupils to dilate. Therefore, an anisocoria that is more prominent in dark indicates a problem with dilatation of affected pupil, suggesting an injury to ipsilateral sympathetic supply to the smaller pupil.

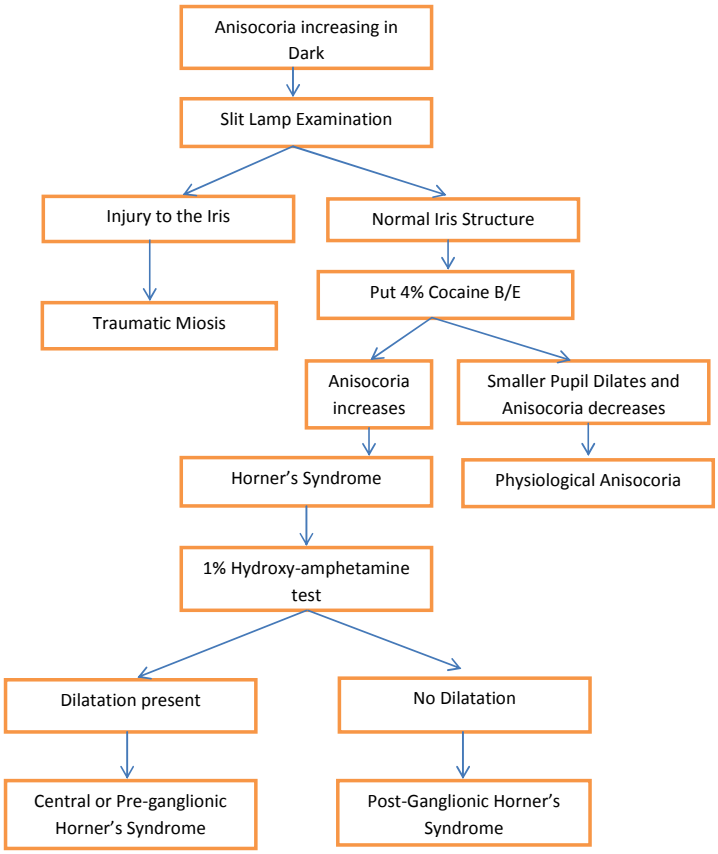
Important conditions causing Anisocoria

Third nerve palsy

One of the most important causes of an enlarged pupil is third nerve palsy. In cases of head injury, expanding supratentorial lesion pushes the uncus against the edge of the tentorium, thereby compressing the underlying midbrain and third nerve. This uncal herniation causes ipsilateral third-nerve palsy and decreased level of consciousness in the patient. These patients require urgent intervention to lower intracranial pressure and carry a poor prognosis if surgical intervention is not done on time.[1] When an anisocoriaco-exists with ptosis and extraocular muscle palsies in a patient with a normal level of consciousness, the diagnosis is third nerve palsy with pupillary involvement.



Flowchart showing work up of case of Anisocoria increasing in Bright light.



Flowchart showing work up of case of Anisocoria increasing in Dark.

In such situations, neuroimaging is indicated to rule out a compressive lesion such as aneurysm of posterior communicating artery which is an emergency or intracranial space occupying lesions.[2] Third nerve palsy due to metabolic diseases, such as diabetes, generally spares the pupil.

Adie's tonic pupil

Adie's tonic pupil is usually unilateral and more commonly found in females aged 20-50 years. Patients may complain of blurred near vision or blurring of vision on changing focus from near to far because of associated accommodation paralysis. The involved pupil shows a poor light response, with a relatively preserved response to near fixation termed as light-near dissociation. The light-near dissociation can be explained by the fact that ratio of fibres controlling pupillary constriction due to near stimulus to fibres controlling pupillary constriction in response to light is 30:1. Therefore, adequate number of fibres remains to preserve the near induced constriction. Some patients may complain of unequal pupils without any other symptoms. Slit lamp examination often reveals sectoral palsy of the iris due to which vermiform movements are present with intact segments contracting in contrast to paralysed segments.

Adie's tonic pupil occurs due to involvement of parasympathetic fibres after they leave the ciliary ganglion or damage to ciliary ganglion. As a result of denervation hypersensitivity, the affected eye displays an abnormally brisk response to dilute pilocarpine (0.125%). Normal eyes generally do not respond to such a dilute solution. This test forms the basis of differentiating preganglionic and postganglionic parasympathetic lesions. [3]

If a patient with tonic pupil has decreased deep tendon reflexes and/or orthostatic hypotension, then it is termed as Holmes-Adie syndrome. The symptoms of a tonic pupil are self-limiting. Adie's pupil is believed to be idiopathic or viral in etiology. Other causes of a tonic pupil include neurosyphilis, diabetes, herpes zoster, giant cell arteritis, and alcoholism. [4]

Horner's syndrome

The disruption of the sympathetic innervations to the eye at any place along the pathway causes Horner's syndrome. Dilation lag is a classic feature of Horner syndrome. Typically, the affected eye has a delayed response to reduced illumination. In patients with a doubtful diagnosis, instillation of 4% cocaine drops is indicated in both the eyes. Cocaine inhibits the nor-epinephrine reuptake causing more norepinephrine to be available at the neuromuscular junction of the iris dilator

muscle. Pupils are assessed after 40-60 minutes. In a positive test, the sympathetically impaired pupil fails to dilate and the degree of anisocoria increases. If both pupils dilate, physiologic anisocoria is the diagnosis. In some studies, this test has been both sensitive and specific for Horner syndrome. [5] 1% Hydroxy-amphetamine test can differentiate central or preganglionic lesions from postganglionic lesions. This becomes important, as patients with postganglionic Horner syndrome tend to have a very good prognosis, while preganglionic lesions are often the hallmark of myelopathy or malignancy.[6] The exception is in patients with carotid dissection, which may result in postganglionic Horner syndrome. However, such lesions also are associated with acute neckpain or other neurologic deficits. Therefore, painful Horner syndromeis an emergency and the evaluation of the anterior cerebral circulation is indicated on urgent basis. In patients with Horner syndrome without pain, a pancoasttumor should be ruled out with a chest x-ray. Horner syndrome also can occur in incipient trans-tentorial herniation which presents with a rapid deterioration in brainstem function and decreased level of consciousness.

Acute angle closure glaucoma

Such cases presents with a painful red eye, diminished vision, nausea, vomiting and signs such as circum-ciliary congestion, corneal edema and shallow anterior chamber with raised intra-ocular pressure. In this condition, the pupil is fixed and mid dilated due to ischemia of sphincter muscle, with an impaired light reflex.

Simple anisocoria

Simple anisocoria may be found in up to 20% of the general population. In most patients, the degree of anisocoria is less than 1 mm, and no ptosis or vasomotor dysfunction is present. In some patients, simple anisocoria may be provoked by oral medications (eg, pseudoephedrine, selective serotonin reuptake inhibitors). Instillation of 4% cocaine solution will dilate the pupil. Old photographs can provide evidence that the anisocoria has been present for some time. [7]

Pharmacologic mydriasis

Pharmacologic mydriasis is one of the causes for dilated pupil. Pilocarpine can be used to diagnose such cases by reversing the pharmacologically induced mydriasis. Pupil size greater than 8 mm and paralysis to both light and near reflex indicates towards a pharmacological dilatation. Patients using scopolamine patches have been noted to have self-limited mydriasis, which has been named as “cruise ship anisocoria.” [8]

Pharmacologic miosis

Pharmacologic miosis can be due to a variety of cholinergic glaucoma medications. A small pupil may be the result of chance exposure to cholinergic agents. In such cases, withdrawal of exposure to the agent confirms the diagnosis.

Bibliography

1. Andrews BT, Pitts LH. Functional recovery after traumatic transtentorial herniation. Neurosurgery. 1991 Aug 1; 29(2):227-31.
2. Fujiwara S, Fujii K, Nishio S, Matsushima T, Fukui M. Oculomotor nerve palsy in patients with cerebral aneurysms. Neurosurgical review. 1989 Apr 1; 12(2): 125-32.
3. Jacobson DM, Olson KA. Influence of pupil size, anisocoria, and ambient light on pilocarpinemiosis: implications for supersensitivity testing. Ophthalmology. 1993 Feb 1; 100(2):275-80.
4. Davis R, Daroff R, Hoyt W. Tonic pupil after temporal arteritis. The Lancet. 1968 Apr 13; 291(7546):822.
5. Kardon RH, Denison CE, Brown CK, Thompson HS. Critical evaluation of the cocaine test in the diagnosis of Horner's syndrome. *Archives of Ophthalmology*. 1990 Mar 1; 108(3):384-7.
6. Wilhelm H, Ochsner H, Kopycziok E, Trauzettel-Klosinski S, Schiefer U, Zrenner E. Horner's syndrome: a retrospective analysis of 90 cases and recommendations for clinical handling. *German journal of ophthalmology*. 1991 Dec; 1(2):96-102.
7. Lam BL, Thompson HS, Corbett JJ. The prevalence of simple anisocoria. *American journal of ophthalmology*. 1987 Jul 1; 104(1):69-73.
8. Riddick FA, Jordan JD. Cruise ship anisocoria. Annals of internal medicine. 1992 Jul 1; 117(1):95.

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