

Anisocoria: A Tale of Two Pupils

Imagine this scene: you're an intern on call in the ICU. You are called to a patient's bedside because the nurse has noticed a "blown pupil." The patient is in his 90s, extremely demented, and uncooperative at baseline. His cooperation level is both due to his dementia, as well as his CO₂ narcosis from respiratory failure; however, the patient is DNI and is essentially dependent on BiPAP. He is reliant on continuous nebulizer treatments. He has a hard time understanding that the mask on his face is what is keeping him alive, so he constantly tries to pull it off of his face. You arrive to the bedside to find an elderly man who will not comply with your neurologic exam. Of course the first thing that was obtained was a CT head without contrast. This returned as having atrophy consistent with the patient's age, but no herniation or bleeds present. Now what?

Anisocoria refers to one's pupils being unequal in size. Causes can range from physiologic to life-threatening. Thus, it is important to distinguish what is the cause of a patient's unequal pupil size. Size of the pupils is governed by two opposing muscle groups in the iris. These are the dilator and sphincter pupillae. The size is predominantly achieved based on the amount of ambient light, but can be influenced by age, emotional state, IOP, and state of arousal.

Constriction of the pupil is mediated by parasympathetic fibers on the **third cranial nerve**.

Dilation of the pupil is mediated by sympathetic fibers originating in the **hypothalamus**.

Examination of the pupil requires a dim room, a bright light source, and a pupillary size gauge. You must note the size, shape, and position of each pupil. The light reflex is tested by shining the light into the patient's eye and observing the magnitude, speed, and symmetry of the direct and consensual response of each eye. The near reflex is only tested if the light reflex is abnormal. This is achieved by having the patient look at a near visual acuity card, usually around one foot from their face. The amount of pupillary constriction should be noted.

In anisocoria, which pupil is the abnormal one? First, a detailed history should be obtained. Is there any history of ocular trauma? Use of topical medications? Exposure to toxins or drugs? Any associated neurologic symptoms? Also, looking at old photographs can be of benefit. Is the anisocoria long-standing? Associated with ptosis? Next, assess the patient's pupils in a dark room. If the **anisocoria worsens in the dark room, the small pupil is the abnormal one**. 0.5-1% apraclonidine can be applied to the small pupil. If it dilates, then this is Horner's syndrome; however, if there is no change, it can safely be said that this is a physiologic anisocoria. If the anisocoria is **more pronounced in the light, then the large pupil is the abnormal one**. If associated with ptosis and ophthalmoplegia, this is a CN III palsy. If the dilation is only finding, 0.1% pilocarpine can be applied to the large pupil. With constriction of the large pupil, a diagnosis of Adie's pupil can be made. If no constriction occurs, 1% pilocarpine should be applied. With minimal or no constriction still, a diagnosis of pharmacologic anisocoria can be made.

Estimates show that **up to 20% of the population has some type of physiologic anisocoria**. Measurements are usually <0.4mm between sides, and there is never a dilation lag. Structural defects are also a common cause, usually causing a change in shape of the pupil. Congenital

defects and numerous acquired ocular conditions can cause anisocoria. Any kind of different sized pupils should have a full ophthalmologic exam, including slit lamp evaluation. Traumatic mydriasis is caused by damage to the pupillary sphincter muscles. Adie's pupil also called a tonic pupil is caused by damage to the ciliary ganglion or short ciliary nerves. This is followed by aberrant innervation. Pharmacologic mydriasis has been seen with several drug classes: topical medications (atropine, homatropine, cyclopentalate, tropicamide, adrenaline, phenylephrine, clonidine, apraclonidine, brimonidine), autonomic drugs (scopolamine patch), aerosolized anticholinergic drugs (ipratropium), and certain plants (jimsonweed).

With the ICU patient, it was a difficult assessment as he was uncooperative with the neurologic exam; however, after a negative CT scan, his anisocoria was thought to be from a non-life threatening condition. The patient was removed from BiPAP for the CT scan and in that short time, his anisocoria resolved (note: pt was receiving ipratropium). The machine that was saving his life was also the cause of his abnormal findings.

References / Further Reading

-“Anisocoria and Horner’s Syndrome.” American Association for Pediatric Ophthalmology and Strabismus. Jul 2013. <<http://www.aapos.org/terms/conditions/27>>

-Mangan, OD. “Anisocoria – Now What?” <<http://www.aaopt.org/sites/default/files/NO-10.pdf>>

-Kedar, MD et al. “Approach to the patient with anisocoria.” UpToDate. Wolters Kluwer Health. 09 Jan 2014. <www.uptodate.com>